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CONTENTS OF PREVIOUS NUMBERS

SEPTEMBER, 1918. NUMBER 3

Immunity Reactions in Hydrated and Concentrated Tissue. F. W. Schlutz, M.D., Minneapolis.

Congenital Atresia of the Esophagus with Report of Four Additional Cases, with Three Necropsies. Joseph Brennemann, M.D., Chicago.

Kala-Azar in a Child (the First American Case). Fritz B. Talbot, M.D., and Arthur B. Lyon, M.D., Boston.

Psychogenic Disturbances in Childhood and Their Treatment. August Strauch, M.D., Chicago.

Cure of Syphilitic Meningitis by Arsphenamin and Mercury. Symptoms—Convulsions and Intense Optic Neuritis—Developed after a Head Injury. Irving M. Snow, M.D., Buffalo.

Head Shaking with Nystagmus in Infants. A Study of Sixty-Four Cases. Charles Herrman, M.D., New York.

The Effect of Injections of Pituitary Solutions on the Urinary Output in a Case of Diabetes Insipidus. S. W. Clausen, M.D., St. Louis.

Current Pediatric Literature.

OCTOBER, 1918. NUMBER 4

Puericulture in France. M. Paul Strauss, Paris, France.

Red Cross Infant Mortality Campaign in France. William Palmer Lucas, M.D., San Francisco.

The Work of the Children's Bureau of the American Red Cross in Lyons. Clifford G. Grulee, M.D., Chicago.

A Public Health Problem in France. C. F. Gelston, M.D., San Francisco.

The American Red Cross in the Meurthe-Et-Moselle. Maynard Ladd, M.D., Boston.

The Massachusetts Child Conservation Committee: The Details of Organization and Work. Richard M. Smith, M.D., Boston.

America's Debt to France. J. H. Mason Knox, Jr., M.D., Baltimore.

A Consideration of the Etiology of Phlyctenular Disease of the Eye. Howard F. Hansell, M.D., Philadelphia.

Is the Amount of Calcium Usually Given in Dilutions of Cow's Milk Injurious to Infants? A. W. Bosworth, A.M., H. L. Bowditch, M.D., and Louise A. Gibbs, B.S., Boston.

The Bearing of Cutaneous Hypersensitiveness on the Pathogenicity of the Bacillus Abortus Bovinus. E. C. Fleischner and K. F. Meyer, San Francisco.

Current Pediatric Literature.

NOVEMBER, 1918. NUMBER 5.

Studies of Infant Feeding. High Protein Feeding Versus High Calcium Absorption as the Cause of the Increase in Body Temperature of Infants. A. W. Bosworth and H. L. Bowditch, M.D., Boston.

A Case with Vascular, Joint, Muscle and Skin Disturbances of Uncertain Etiology. Richard M. Smith, M.D., and Warren R. Sisson, M.D., Boston.

The Effect of Alkali and Malt Preparations on the Retention of Calcium in Infancy. Akira Sato, M.D., Japan.

Migraine in Childhood. Wilder Tileston, M.D., New Haven.

The Relative Efficiency of the Different Mercurial Preparations in the Treatment of Congenital Syphilis in Infants and Children. Walter Reeve Ramsey, M.D., St. Paul, and Mildred R. Ziegler, M.S., Minneapolis.

Progress in Pediatrics. Recent Work in Anatomy, Physiology and Pathology of Childhood. James B. Holmes, M.D., Baltimore.

Current Pediatric Literature.

DECEMBER, 1918. NUMBER 6.

Standards for Growth and Nutrition. L. Emmett Holt, M.D., New York.

Experimental Research on the Excretion of Poisons. Naoyoshi Okubo, M.D., Osaka, Japan.

A New Apparatus for Puncture of the Superior Longitudinal Sinus. Alton Goldbloom, M.D., New York.

Tertian Malaria in Two Infants Under Two Months of Age. Charles James Bloom, M.D., New Orleans.

Progress in Pediatrics:
Recent Work in Anatomy, Pathology and Physiology of Childhood. James B. Holmes, M.D., Baltimore.

Current Pediatric Literature.

Index to Volume XVI.

JANUARY, 1919. NUMBER 1.

Studies in the Adaptation of an Artificial Food to Human Milk. II. A Report of Three Years' Clinical Experience with the Feeding of S. M. A. (Synthetic Milk Adapted). H. J. Gerstenberger, M.D., and H. O. Reik, M.D., Cleveland.

A Method for the Determination of Fat in Dried Feces and Its Distribution as Soap, Free Fatty Acids and Neutral Fat. An Application to Feces of the Roese-Gottlieb Method for Determining Fat in Dried and Condensed Milks. L. Emmett Holt, M.D., Angelia M. Courtney and Helen L. Fales, New York.

Clinical Department:
Observations on a Case of Rumination with Suggestions as to Treatment. M. D. Batchelor, M.D., and R. P. Batchelor, M.D., Palmerton, Pa.

A Case of Cardiospasm, Followed by Hypertrophy, Dilatation and Stricture of the Esophagus, Treated by Gastrostomy, and Subsequent Dilatation of the Esophageal Stricture. E. E. Graham, M.D., Philadelphia.

Progress in Pediatrics:
Review of Literature on Respiratory Diseases. Louis C. Schroeder, M.D., New York.

Current Pediatric Literature.

FEBRUARY, 1919. NUMBER 2

Comparison of the Carbon Dioxid Tension of the Alveolar Air and the Hydrogen-ion Concentration of the Urine with the Bicarbonate of the Blood Plasma. O. M. Schloss, M.D., New York, and Helen Harrington, M.A., Baltimore.

Fetal Bronchiectasis. H. L. Koeckert, A.B., M.D., Cleveland.

Study of the Intestinal Contents of Newly Born Infants. A. Hymanson, M.D., and Max Kahn, M.D., Ph.D., New York.

Sleep in Children and Its Disturbances. A. Strauch, M.D., Chicago.

Progress in Pediatrics:
A Review of the Literature of the Past Year (1918) on Genito-Urinary Diseases in Children. Lewis Webb Hill, M.D., Boston.

Current Pediatric Literature.

American Journal of Diseases of Children

Vol. 17 MARCH, 1919 No. 3

EPIDEMIC INFLUENZA IN INFANTS*

MARTHA WOLLSTEIN, M.D., AND ALTON GOLDBLOOM, M.D.
NEW YORK

When the present pandemic of influenza made its appearance along the Atlantic seaboard there was a general impression that children were not affected by the disease. This may have been justified at the very outset when the epidemic was limited to the military camps. As soon as the disease spread to the civilian population, however, cases among children of all ages became numerous. Thus, Dr. Hatfield[1] found that there were 50 subjects under 5 years of age among 339 patients treated in the contagious hospitals of New York City, or an incidence of 16 per cent. At the Babies' Hospital infants ill with influenza came for admission during the last week in September. From Oct. 1 to Nov. 15, 1918, 36 cases of "bronchopneumonia with influenza" were admitted, while during the same six weeks in 1917 only seven such cases presented themselves. This year (1918) 52 children came into the hospital with pneumonia between October 1 and November 15. Last year (1917) thirty-eight children with pneumonia were admitted during the same period; an increase of 14 cases, or of 37 per cent. The real significance of this increase, however, is shown by the mortality rate, which was 15.8 per cent. (6 cases out of 38) in 1917, and 57.7 per cent. (30 out of 52) in 1918.

The comparative incidence in mortality of all types of pneumonia admitted to the hospital during the same period of six weeks in 1917 and 1918 is shown graphically in the accompanying charts (Figs. 1 to 6, inclusive).

Narrowing down the pneumonia cases to those diagnosed as pneumonia with influenza, we find that there were seven such patients last year (1917), all of whom recovered. This year (1918), of 36 children who had bronchopneumonia and influenza, twelve recovered and twenty-four died, a mortality rate of 66.6 per cent. Of the 24 fatal cases, 18 came to necropsy and form the basis of this study.

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* From the Babies' Hospital of the City of New York.

1. Hatfield, Hazel M.: Paper read at the meeting of the Woman's Medical Society of New York, Nov. 20, 1918.

The age of the patients ranged from 5 weeks to 21 months. Four children were less than 6 months old, 9 were under 1 year and 9 were between 1 and 2 years of age. There were 7 girls and 11 boys.

In nine cases no history of family contagion could be obtained. On the other hand, the mother had influenza in two instances, with fatal result in 1; the father, in another; two other children were ill in one family, and in three families one other child had the disease. Thus direct exposure to family infection was evident in seven of the little patients. Two children who had been in the hospital for several weeks with colitis were exposed to influenza there.

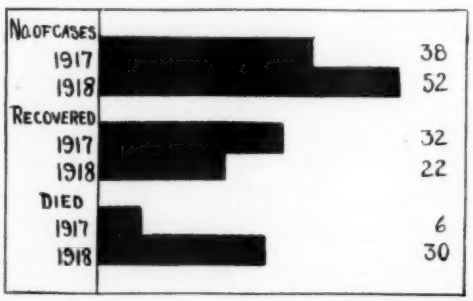

Fig. 1.—All pneumonias, October 1 to November 15.

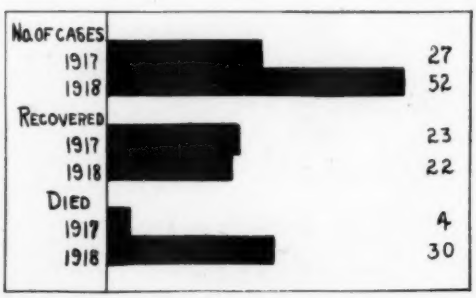

Fig. 2.—Bronchopneumonias, October 1 to November 15.

Since these patients were not brought for admission until they had developed pneumonia, it is evident that the clinical symptoms of the early, uncomplicated stage of influenza in young children cannot be elucidated by a study of our series. Even the two cases which developed in the house were masked by the original disease of the patients. The temperature showed daily remissions of several degrees and was irregular; it ranged between 107 and 96 F. It became subnormal before death in some cases and rose before the fatal termination in others.

Cyanosis was marked in 3 cases, moderate in 5, absent in 8, and not noted in 2. Marked general redness of the throat was noted 9 times; 7 children had normal throats, and 2 were moribund on admission so that no examination was made.

In the fatal cases the leukocyte count varied. In 10 it was over 20,000. The highest number of white cells was 40,000. In 5 other cases the leukocytes varied from 12,000 to 17,000, which is scarcely above normal for children under 2 years old. One syphilitic infant

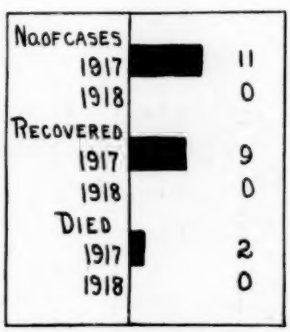

Fig. 3.—Lobar pneumonias, October 1 to November 15.

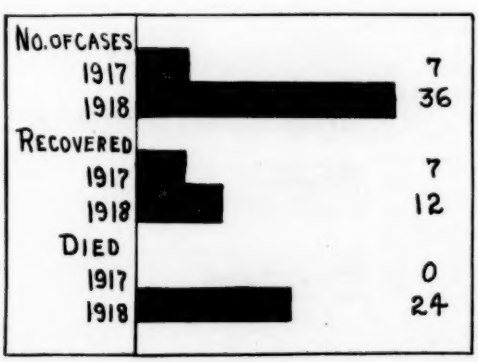

Fig. 4.—Bronchopneumonia with influenza, October 1 to November 15.

had only 8,400 white cells with 82 per cent. mononuclears. In the other cases the mononuclear cells varied from 22 to 58 per cent. The polynuclear leukocytes reached 78 per cent. and 71 per cent., respectively, in 2 cases of lobar pneumonia; and 72 per cent. in a case of bronchopneumonia of no greater severity or extent than 6 others in which the white cells were not above 60 per cent. The only patient who did not present any consolidated lung tissue at necropsy had had 34,000 leukocytes with 66 per cent. polynuclears, during life.

In no other instance was a count obtainable early in the disease, before the onset of complicating pneumonia. It may be said, however, that in this series of 18 cases a definite leukocytosis was found in 10, no increase occurred in 5 and a leukopenia was present in 1 syphilitic infant. Two children were admitted in a moribund condition and no blood examination was made. The leukocytosis was accompanied in every instance but one by a pneumonia. One patient without pneumonia had a white cell count of 34,000, and 5 patients

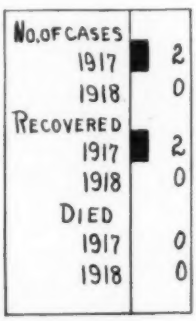

Fig. 5.—Lobar pneumonias with influenza, October 1 to November 15.

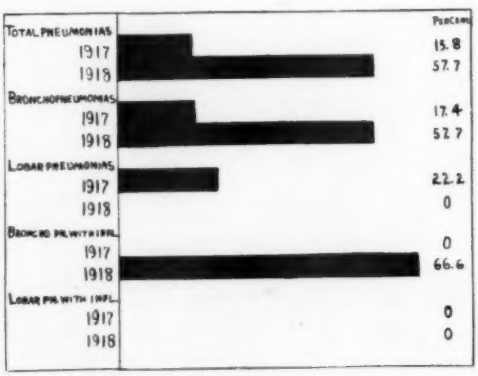

Fig. 6.—Mortality, per cent.

with pneumonia had no rise in leukocytes. In other words, there was no definite blood picture in this series.

The average duration of the illness was seven days in these fatal cases; the shortest was four days and the longest, twenty-five days. Owing to the indefinite history given on admission, these figures cannot be accurately determined.

The complicating conditions which were present in those children who died of influenzal pneumonia were varied. One child showed

marked lesions of congenital syphilis; another had general miliary tuberculosis; 4 infants had ulcerative colitis, complicated in one by pyelitis; four were markedly rachitic. All these complications simply predisposed the patients to infection and were present before the fatal illness began. One baby developed pneumococcus meningitis before death, while two with ulcerative colitis and one with septic peritonitis developed marked abdominal purpura before the fatal termination.

BACTERIOLOGY

Cultures were made from the sputum before death in 17 of the 18 patients. *B. influenzae* was present 13 times: in overwhelming numbers, once; very numerous in 11; few in 1. Five times the organism was not found in the sputum, probably because of the difficulty of obtaining suitable specimens for culture from the prostrated patients. The other bacteria present were pneumococci, staphylococci, *Micrococcus catarrhalis* and streptococci, in the order of frequency. Only Type IV pneumococcus was found.

Eighteen cases came to necropsy. In all, the influenza bacillus was cultivated from both lungs in large numbers, but never in pure culture. It was found once in the heart's blood, together with the pneumococcus, and once in pus from an empyema. The only case of meningitis which developed among these children was due to pneumococci, and influenza bacilli were not present in the spinal fluid. The heart's blood was sterile in 8 cases; it contained the pneumococcus in 5. Pneumococci were grown from the lungs in 14 cases; streptococci once and *M. catarrhalis* 4 times. Twice *M. catarrhalis* was the only other organism found besides *B. influenzae*. Morphologically and biologically the influenza bacillus isolated from these children did not differ from the respiratory strains isolated from endemic cases in other years. It was a short, pleomorphic, gram-negative bacillus, strictly hemoglobinophilic, growing in characteristic dewdrop colonies on blood agar. On Dr. Avery's[2] oleate agar the colonies were larger and grayish. On animal inoculation the organisms also conformed to the respiratory strains, in that they were not pathogenic for rabbits or guinea-pigs; but they were less virulent for mice than is usually the case with strains of influenza bacilli isolated from the respiratory tract.[3] The absence of the bacilli from the heart's blood except in one case, and the fact that no meningeal infections occurred are also in keeping with the low virulence of the organisms for animals. On the other hand, the failure of animal inoculation may be due to the highly "humanized" condition

2. Avery, O. T.: J. A. M. A. **71**:2050, 1918.
3. Wollstein, Martha: J. Exper. M. **22**:445, 1915.

of the bacillus. It is certain that it is highly invasive for every part of the respiratory tract, though it enters the blood stream so rarely.

In this country, Dr. Anna W. Williams[4] found influenza bacilli in heart blood cultures made at necropsy 3 times among 30 cases studied. The commission reporting on influenza among British soldiers in France found the bacillus in the blood in 2 of 68 cases cultured during the acute febrile stage of the disease.[5] Influenzal meningitis occurred in only 2 cases among the English soldiers. The low incidence of meningeal involvement is remarkable and points to an absence of meningeal strains of the influenza bacillus from this pandemic. The low percentage of positive blood invasion is also in keeping with the low average virulence of the respiratory strains of *B. influenzae*. This tends to substantiate the view that influenza alone is a disease which tends towards recovery in the majority of cases, and that the fatal issue is due, except in the comparatively rare instances of highly virulent respiratory strains of the influenza bacillus, to complicating secondary infections of the lungs with other organisms, chiefly pneumococci and streptococci. The meningeal strains, on the other hand, are almost invariably highly invasive for the blood stream and cause a suppurative leptomeningitis which is practically always fatal. We have, as yet, no accurate knowledge of the nature of the toxin produced by both meningeal and respiratory strains of *B. influenzae*.

PATHOLOGY

The necropsy findings on these 18 cases were very similar. The interest centered in the lungs, which contained a great variety of lesions in every case. Pleural exudate was present in 13 of the children; in 9 it was only an exudate of fibrin over the consolidated areas, varying from a thin layer to a sheet 2 mm. in thickness. In 4 cases fluid pus was present as well. The color of the lungs was bright red, due in part to intense congestion, in part to many subpleural hemorrhages (Fig. 7). Bronchopneumonia was present in 17 of 18 cases, accompanied by lobar pneumonia in 2, multiple lung abscesses in 2, and general mucopurulent bronchitis in every one of the 18 cases. Pulmonary edema was marked in 12 instances. A superficial emphysema was noted several times, over all the lobes. In none of the 17 cases with pneumonia was only one lobe involved. All lobes were involved in 3 instances; four lobes in 8, three lobes in 5 and two lobes in 1 case. Thus, in 16 of the 18 cases more than two lobes were involved. The

4. Williams, Anna W.: Paper read at the meeting of the Woman's Medical Society of New York, Nov. 20, 1918.

5. Influenza Committee of the Advisory Board to the D. G. M. S., France, Brit. M. J. **2**:505, 1918.

character of the lesions varied greatly. The extent and migratory character of the pneumonia was shown by the fact that the lesions in the several lobes were not of the same age, but varied from small areas of peribronchitis to larger conglomerate areas occupying all or most of the lobe. Purulent infiltration was very marked, and abscesses

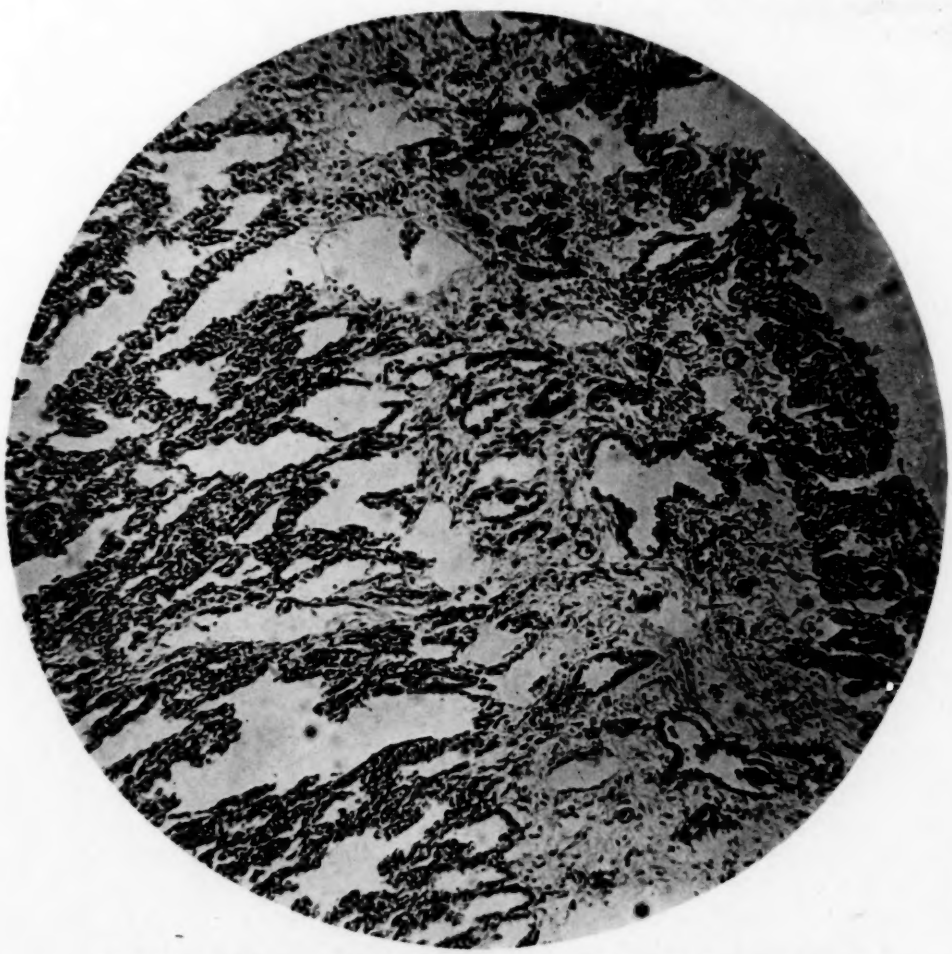

Fig. 7.—Section of lung showing congestion of the blood vessels in the pleura and hemorrhages just beneath the pleural surface.

had formed in two cases. Lobar pneumonia in the stage of gray hepatization was present in the right lower lobe in 1 case, while bronchopneumonia with hemorrhages was found in the other two lobes of the same lung and in one lobe of the left lung. The bronchitis was general, even in the lobes which were not involved in the pneumonic process.

There were subcapsular hemorrhages over the thymus, thyroid, liver, kidneys, suprarenals and lymph nodes; submucous hemorrhages in the larynx, trachea, stomach, intestines and kidney pelvis. The bronchial and mediastinal lymph nodes were swollen, edematous, red in color, often very deep red; and on section, small hemorrhages were

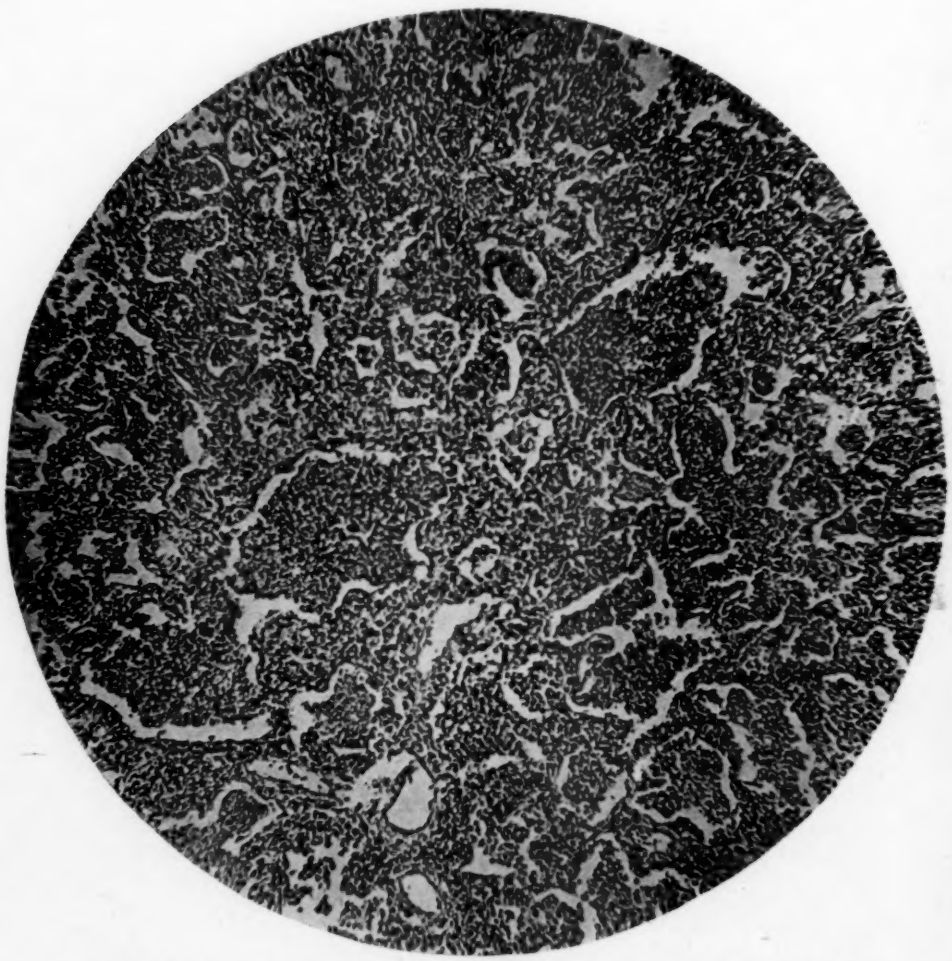

Fig. 8.—Section of lung showing suppurative bronchitis and areas of pneumonia about the bronchi. The exudate is chiefly polynuclear in character.

visible, usually one or two in a node. The spleen was congested; acute splenic tumor was present twice and chronic hyperplasia 6 times, due to syphilis in 1 and rachitis in 4. The liver showed no characteristic lesion. The kidneys were very much congested and showed marked parenchymatous degeneration, with punctate hemorrhages

beneath the lining of the pelvis, and about the apexes of the pyramids. The suprarenals were not enlarged or unduly congested. Subendocardial hemorrhages were noted once; petechial hemorrhages into the skin were present 3 times; but all these 3 cases were complicated, by ulcerative colitis in 2 and by septic peritonitis (*Staphyloccus pyogenes aureus*) in one.

On microscopic examination the lungs showed thrombi in many vessels, with hemorrhages into the alveoli. Edema of the vessel walls and connective tissue septa was very marked. The bronchi contained polynuclear cells and red blood cells; their walls were densely infiltrated and surrounded by alveoli packed with cells; chiefly polynuclear (Fig. 8). The alveolar contents were poor in fibrin, except in the lobar pneumonia lesions. The most marked and characteristic features were purulent infiltration of the alveolar walls and septa, hemorrhages and suppurative bronchiolitis. The capillaries and all the large blood vessels were distended with blood. Masses of bacteria were demonstrable in the bronchi and alveoli in one case, but with Gram's stain these were found to be cocci which retained the stain.

The other organs showed nothing characteristic on microscopic examination.

SUMMARY

To sum up: our eighteen cases showed a variety of pulmonary lesions making a characteristic picture of congestion, edema, general bronchitis, scattered pneumonia lesions differing in age, but involving two or more lobes; subpleural hemorrhages and pleural exudate. Capillary hemorrhages in the other viscera were constant. The influenza bacillus was grown from every case, but not alone. Pneumococcus Type IV was the most frequent bacterium associated with it. During life the sputum contained *B. influenza* in thirteen of seventeen cases in which it was searched for. This series of pneumonias was far more severe, more extensive and more highly fatal than was any other group of pneumonia cases observed in the hospital over a similar period of time.

REPORT ON AN UNUSUAL CASE OF APLASTIC ANEMIA*

LAWRENCE WELD SMITH, A.B.
BOSTON

An unusually interesting case of anemia came under observation during the past summer at the Children's Hospital. The results of the rather careful study of the case are presented with the hope that they may be of some value in clearing up the uncertainties which unfortunately surround many of these vague blood conditions. The following is the clinical history in brief, touching on all the points which seem of the slightest diagnostic significance in considering the case.

REPORT OF CASE

J. K., aged 6 years and 3 months, was admitted April 6.

Birthplace, Middleboro, Mass.; nationality, Russian.

Family History.—The father has been told his left lung is diseased. No family history of syphilis, hemophilia, anemia, etc. Two brothers are living and well.

Personal History.—Full term, normal delivery; breast fed for six months; measles eight months and summer complaint eighteen months prior to admission; subject to colds and sore throat.

Present Illness.—General malaise for the previous two weeks; anorexia; has seemed very weak. His rest has been disturbed by dreams and hallucinations of sight. This latter condition may have been present somewhat for three months. Bowels irregular lately. Urine has been examined and reported negative.

Physical Examination.—Child lying quietly on the table; rather apathetic and cachectic in appearance, with a peculiar sallow complexion, and marked pigmentation, particularly of the upper eyelids. Eyes are dreamy. He seems to be conscious, but very inanimate. Head, eyes, ears, nose, throat, all negative.

The neck shows a few pea-sized palpable glands.

The thorax is rather flat; no rachitic evidence; sits in a slumping position, with the spine arched.

Heart: Apex diffuse; felt in fourth and fifth spaces; measures 2 by 7 cm.; systolic murmur heard over whole precordia, transmitted to axilla; action regular (124), rapid; A2 and P2 normal.

Lungs: Dulness on percussion over left apex, with impaired vocal resonance posteriorly; no râles heard.

Abdomen markedly sunken; liver edge just palpable; otherwise negative. Extremities and reflexes all normal.

Skin poorly nourished; dry, sallow, with a peculiar grayish-yellow color suggestive of Addison's disease or pernicious anemia.

Extracts from House Records: April 7: Von Pirquet positive after thirty-six hours for both human and bovine types. Urine, negative. Blood: Red cells, 1,514,000; white cells, 6,200; hemoglobin (Sahli), 40 per cent. Differential Count: Polymorphonuclears, 49 per cent.; lymphocytes, 41 per cent.;

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* From the Medical and Pathological Departments of the Children's Hospital.

basophils, 2 per cent.; myelocytes, 8 per cent. The red cells show some poikilocytosis, anisocytosis and polychromatophilia. There are a few cells which resemble myelocytes, as indicated in the foregoing, and numerous cells which are probably normoblasts. The platelets are about normal in number although possibly somewhat decreased.

April 8, 1918. Feces, negative; show no indigestion of any of the food elements, starch, fat or protein; no ova or parasites seen. Roentgen Ray: Lungs show nothing pathologic.

Treatment.—Iron citrate in ampoule form was given subcutaneously every three or four days.

Diagnosis.—A diagnosis of secondary anemia and mitral regurgitation was made.

Result.—The patient improved noticeably under treatment and observation and was discharged April 26, 1918.

Readmission, July 6, 1918.

Present Illness.—Following discharge home the child was much better for two weeks, and since then has been steadily going down hill. The bowels are constipated; periods of hallucinations increasing, always at night; father has noticed periods of swelling of the lips, nose, head and neck. About a week ago there was a swelling over the forehead over an inch in thickness. It was nonpainful, did not pit on pressure, and disappeared within an hour. His cough has increased. The patient has never been out of Massachusetts; anemia not likely, therefore, to be due to any parasitic infection.

Physical Examination.—Patient is a thin, yellowish-brown pigmented boy, aged about 7 years. The pigmentation is most marked on the back of the neck, the genitals, the umbilicus and the areolae. In general the physical examination is the same as on previous admission. The heart, however, is possibly somewhat enlarged. It measures 2 by 7.5 cm. The impulse is felt outside the nipple line, and is not heaving in nature. The sounds are regular. There is a rough systolic murmur with its point of maximum intensity over the apex, but heard over the entire precordia and transmitted to the axilla. P2 is doubled and accentuated.

Laboratory Findings: July 6: Blood: red blood cells, 762,000; white blood cells, 4,900; hemoglobin (Sahli), 18 per cent.; blood pressure, 115-40. Differential Count: polymorphonuclears, 25 per cent.; lymphocytes, 67 per cent.; large mononuclears, 3 per cent.; eosinophils, 1 per cent.; myelocytes (?), 4 per cent. Smear shows well marked achromia, moderate poikilocytosis, and slight anisocytosis. The lymphocytes are chiefly of the small type. No normoblasts are seen; no plasmodiae noted; no stippling or polychromatophilia.

July 7. Urine, negative; feces, negative.

July 10. Heart measures 3 by 8 cm.; action regular; slight systolic murmur over whole precordia; slight diastolic murmur at apex; P2 greater than A2; pistol shot and systolic murmur heard in groin (Morse).

July 11. Wassermann negative.

July 12. Up in clothes; feeling better; iron treatment being given.

July 16. Rise in temperature; purpuric areas on front of both thighs.

July 23. Swelling of upper lip without pain or redness; subsided rapidly.

July 24. Blood: red blood cells, 788,000; white blood cells, 3,600; red cells show marked anisocytosis, poikilocytosis and achromia; no nucleated reds seen; no polychromatophilia. Differential Count: Polymorphonuclears, 15 per cent.; mononuclears (mostly lymphocytes), 81 per cent.; transitionals (in previous smear, thought to be myelocytes), 4 per cent.

July 27. Sudden rise in temperature to 105 F., for no apparent cause; ears aching; drums congested and dull; abdomen distended and tympanitic; headache; puffiness about lower lids; somewhat delirious.

July 29. Somewhat better; still has puffiness of face.

July 31. Slight swelling of left cheek; ulceration at junction of buccal membrane and gum.

August 1. Swelling and ulceration both increased.

August 5. Urine contains a few leukocytes. Blood: red blood cells, 408,000; white blood cells, 5,400.

August 13. Temperature, pulse and respiration all increased. The patient looks poorly, feels very poorly; cough increased; complains of a substernal pain; lungs clear, and no râles heard.

August 15. Blood: red blood cells, 408,000; white blood cells, 4,000; hemoglobin, 10 per cent.

August 17. Cough increased, severe; complains of pain in left ear on coughing; evidently much worse; feels very poorly.

August 18. Severe precordial pain; vomited once.

August 19. Pain much more severe; given morphin (1/16 grain per orem). During the day became progressively worse; exhausted; respiration became gasping, and he sank gradually, dying from heart failure at 1:25 a. m., Aug. 20, 1918.

CLINICAL DISCUSSION

In brief, the clinical history can be summed up as follows: a boy of 6 who has been apparently well up to the time of the present illness, with the exception of measles eight months previously, comes into the hospital with a history of only two weeks' illness, with no definite symptoms. The clinical picture and the laboratory findings are roughly those of a rather severe secondary anemia, cause unknown. There had been no history of hemorrhage, or of any more recent infection than the measles. He responds temporarily to iron treatment, but soon becomes more anemic and progresses rapidly downhill to fatal exitus. There are very slight purpuric manifestations, and an unexplained swelling of the face, accompanied by high fever. The blood picture, at first suggestive of secondary anemia, becomes more definitely that of an aplastic anemia.

Let us consider the story for a minute from this, the clinical side, before the pathologic evidence is introduced. Gruner's work in 1914 on the biology of the blood cells is perhaps the most satisfactory conception, and more satisfactorily classifies the blood disorders by reference to their point of origin than any of the other modern investigators. In these obscure anemias a diagnosis can only be made by a very careful study of the blood picture, and thorough knowledge of the development of the blood cells is an invaluable aid.

Let us turn to the original blood picture on admission. The other superficial signs of anemia are of little value in differential diagnosis. The pallor, the pigmentation, the anorexia, the disturbed sleep, the irregular bowels, the weakness all fit in equally well with any severe anemia, with the possible exception of the pigmentation, and that we can leave for the moment unexplained. The degree of anemia even at this time is extreme. A hemoglobin of 40 per cent. and a red count of a million and a half in a boy of 6 is seldom noted with a history of

only two weeks' duration. The differential white count shows a beginning relative lymphocytosis and a drop in the leukocytic proportion. In a child of 6 this is less definite than it would be in an adult. The presence of a good deal of poikilocytosis, anisocytosis and polychromatophilia is, on the other hand, of very great diagnostic value usually, especially when this is further supported by the presence of numerous normoblasts and possibly a few myelocytes. It is a definite indication of marrow activity, or at least of activity of the hematopoietic organs. It is to be regretted that a few other laboratory tests were not carried out — the vital staining of the red cells, the test for fragility in hypertonic salt solution, the bleeding and coagulation times, the retractility of the clot, etc. — but they would have been of interest rather than of any great value in this particular case. The diagnosis of probable secondary anemia was made at this time chiefly because of the active regeneration of the marrow as evidenced by the rather characteristic blood picture. A primary anemia was considered, but with the lack of megaloblasts and macrocytosis, it was dismissed rather summarily, especially as under the iron treatment and general hygiene he improved so very markedly.

During the next two months he was lost sight of, but on readmission, in an even weaker state than previously, and obviously more anemic, the rather obvious primary anemia diagnosis was uppermost in mind. That this was very probable was borne out by the laboratory findings again. A red count of 700,000, a hemoglobin of only 18 per cent., and an obvious relative lymphocytosis could be accounted for most readily by such a diagnosis, even in the absence of normoblasts. At this time little else could be thought of as a differential. Purpura had to be excluded for there was no evidence of hemorrhage anywhere from any of the mucous membranes or into the skin. There was the differentiation between primary pernicious and primary aplastic anemia to be considered, and other than that there was nothing but that rather vague and indefinite group of atypical purpuras with angioneurotic edema, abdominal pain, etc., which Osler mentioned, and the possibility of an aleukemic phase in a case of an acute leukemia. There was nothing to do but follow the case and watch the changes in the blood picture to confirm the diagnosis. This was accordingly done. Red and white counts were made at frequent intervals, differentials done and every care taken to follow the changes in the blood. Thus an aleukemic phase of a leukemia was easily excluded. Again, a primary pernicious anemia never persists with the absolute lack of marrow activity, so far as we know, which this case exhibited, as demonstrated in the peripheral circulation by the lack of young cell forms.

Thus, the diagnosis comes down either to aplastic anemia or one of those cases of atypical purpura described by Osler. Purpuric-like petechiae did appear in the thighs. Angioneurotic edema of the face occurred. These were both late symptoms, sufficiently so, it seems, to exclude purpura as a diagnosis. In support of the aplastic anemia diagnosis, then, we have the following points, which seem fairly conclusive from the clinical point of view:

1. The age of the patient — 6 years — which almost automatically excludes primary pernicious anemia. *

2. The fact that the blood pictures never definitely developed into the characteristic pernicious anemia picture.

3. The rapid course, the onset apparently within two weeks, and the entire clinical course occupying less than five months.

4. The cachetic appearance and possibly the pigmentation.

5. The rather characteristic blood pictures toward the latter part of the disease.

6. The purpuric petechiae, the angioneurotic edema, the fever without obvious infection; all more or less characteristic of aplastic anemia. This, then, is the evidence on which the diagnosis of aplastic anemia was made in this case, and which points rather definitely to the idiopathic nature of the disease. The use of the word idiopathic perhaps calls for an explanation. By it is simply meant in this instance a blood disease with a perfectly definite blood picture, which is apparently primary in nature, or at least which has no obvious cause as a secondary anemia following hemorrhage. We will postpone for the moment the discussion of the relation of this disease to the purpuras or to the other anemias and accept its idiopathic nature and its clinical entity. There is, of course much discussion as to whether there is even such a disease, and if so, whether it is a distinct entity or merely a subdivision of the other primary anemias. And if we accept its existence there is then the much debated point as to whether the aplasia of the marrow is the primary pathologic lesion, or whether the aplasia is secondary to some toxic, possibly hemolytic, agent acting in the peripheral circulation.

Before we consider the case any further, let us pass on to the pathologic findings at necropsy in this case. I will only include those parts of the necropsy which are of interest or of diagnostic value and will dismiss the rest of the findings as briefly as possible. Most of the interest is centered in the bone marrow, the liver, the spleen, the heart and the lungs.

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* Morse and Wolbach: Case of Pernicious Anemia in a Boy of Eight, Am. J. Dis. Child. **14**:301 (Oct.) 1917.

Pathologic Findings.—Body: The body is that of a fairly well developed and poorly nourished child, measuring 117 cm. in length. The skin has a marked pallor, but is deeply pigmented, especially on the exposed surfaces, the axillae, the groins and genitalia. The mucous membranes are pale, the conjunctivae practically bloodless.

Peritoneal Cavity: There is almost no fat under the skin or in the abdominal cavity, the mesentery and omentum being nearly devoid of any fat tissue. What fat is present is normal in color. The abdominal viscera are all very pale. The lymph nodes (mesenteric) are not enlarged. The diaphragm is normal. There is no free fluid.

Pleural Cavities: Each pleural cavity contains about 50 c.c. of slightly blood-tinged fluid (blood probably from necropsy incision).

Pericardial Cavity: The pericardial cavity contains about 50 c.c. of a clear straw colored fluid.

Heart: The heart weighs 175 gm. It is enlarged, dilated and pale. Under the epicardium there are numerous petechial hemorrhages varying in size from a pinpoint to over 2 mm. On cutting the great vessels of the heart, thin watery blood, apparently under some tension, gushes forth. There are no clots in the great vessels or in the left side of the heart. In the right auricle and its appendage there are a few small dark red elastic recent clots. Under the endocardium of the right ventricle and auricle there are similar petechiae, less numerous. The myocardium presents a markedly mottled appearance, due to transverse opaque yellow lines, especially over the papillary muscles (typical tigroid mottling). The myocardium is extremely pale. A few of the chordae tendinae of the posterior group of papillary muscles appear to be somewhat thickened and shortened. The valves are normal, except the mitral, on the posterior segment of which, near the valve edge, are three small pinhead sized, elevated, pigmented, brownish areas resembling fresh vegetations. The valves measure: tricuspid, 8 cm.; pulmonic, 5 cm.; mitral, 7.5 cm.; aortic, 4.5 cm. The right ventricular wall measures 4 mm.; the left wall measures 7 mm. in thickness.

Microscopic: The cross section shows a very mottled appearance of degenerated muscle fibers and normal muscle fibers. The degeneration consists of fatty infiltration which is everywhere prominent. The fat is found in drops of varying size, many fairly large, which by special stains is found to be chiefly neutral fat, although some of it is in the form of lipoids. The nuclei show little or no changes. Marked myocardial degeneration with fatty infiltration sums up the situation.

Lungs: The lungs do not collapse on opening the chest. The right lung weighs 250 gm.; the left 190 gm. On the surface of the lungs are several small dark reddish areas about pinhead in size. On the posterior surfaces of both lungs are several larger reddish purple blotches covering one to several lobules. Interspersed with these are a few smaller dark red areas about 0.5 cm. in diameter. These latter feel firm to the touch and are not crepitant like the rest of the lungs. With the exception of these areas the lungs are crepitant throughout. On section of the lung tissue, especially of the right, there exudes a clear serous fluid mixed with air bubbles. There are a few discrete areas of bronchopneumonic spots, dark red with a granular surface and firm to the touch, scattered throughout the posterior portion of both lungs. The vessels are normal. The bronchi are normal. The interlobular lymphatics stand out very prominently.

Microscopic: The lungs for the most part are normal. Areas of bronchopneumonia are present, but they lack the true appearance of a pneumonia, for there is comparatively little leukocytic infiltration. The type cell is the wandering mononuclear leukocyte, although there are some polymorphonuclear cells. The exudate is chiefly serum and fibrin, however. The edema is very noticeable. There are places where the exudate is hemorrhagic in nature. The red cells show acidophilic stippling. A few alveoli have their epithelium completely lacking, but in general there is very little destruction to the lung tissue.

Liver: The liver weighs 440 gm. The surface is smooth and the capsule glistening. There is a marked pallor with a distinct brownish tinge. The cut surface is smooth, moist and of a peculiar golden-brown color. The tissue is fairly firm. It is more opaque than usual. The centers of the lobules appear pale and rather suggestive of fatty degeneration. There is a very definite suggestion of hemosiderin deposit from the peculiar color. The vessels appear normal. The lymphatics are rather prominent.

Microscopic: The liver shows very marked central necrosis, with fatty degeneration and infiltration. Peripherally in the lobules there is tremendous hemosiderin deposit. This is best seen by the potassium ferricyanid blue reaction. The liver cells are undergoing marked necrosis centrally. The nuclei show both karyolysis and pyknosis, the former being very suggestive of glycogen deposit. The cytoplasm of the central liver cells is filled with fat droplets of varying size as seen with Scharlach R and Nile blue sulphate stained specimens. The pigment deposit is very much more marked peripherally in the lobules. It is in rather fine granules and arranged chiefly around the nucleus. The sinusoidal epithelium appears to be normal, although rather distended in places as though edematous. Blood cells are very few in number. The red cells show the acidophilic stippling mentioned previously. In the central veins there are accumulations of fibrin and degenerated red cells. In the portal areas the vessels and the bile ducts appear normal. The lymphatics are all distended and filled with coagulated lymph and what appears to be fibrin (Figs. 1 and 2).

Spleen: The spleen is very small and firm, but rather lax. It weighs 18 gm. The surface is practically smooth. The capsule is a little thickened. It is bluish red in color. On section the trabeculae are very prominent and very close together. There is increased resistance to the knife on sectioning the organ. The malpighian corpuscles cannot be distinguished. On section the color is dark red.

Microscopic: The capsule is a little thickened. There is an increase of connective tissue throughout the organ, the trabeculae running in from the capsule, being unusually thickened and prominent. The malpighian corpuscles are the striking feature of the histology. They are very prominent and numerous. They appear to be rather smaller than normal and almost coalescent, there is so little splenic pulp. The type cell is the large lymphocyte which shows a tendency to be polygonal. There are numerous rather larger cells which resemble plasma cells. The centers of the corpuscles are undergoing a very definite necrosis. There are numerous pyknotic polymorphonuclear cells there, a good deal of fibrin and considerable fat deposit, chiefly in the mononuclear cells. These are more or less intimately fused, forming what appear to be large giant cells, reminiscent of tubercles, rather typical of toxic degeneration (Mallory's Textbook of Pathology). (Fig. 3.) There is evident edema, for the cells are very widely separated. The pulp reticulum can be clearly seen. In the meshes are a few cells, but the pulp is very limited in amount. The cells are mostly large lymphocytes, plasma cells, and a very few achromatic reds, many of which are stippled as mentioned before. There is no evidence of cellular activity in the spleen as far as blood formation is concerned.

Bone Marrow: The ribs are small in size. They are very pale and do not show the red pulp shining characteristically through them. On section only a very small amount of pale yellow fatty substance can be squeezed out. The marrow in the upper third of the femur is uniformly pale yellow in color, and fatty. There is the most complete aplasia present.

Microscopic: The bone marrow from the femur shows practically no cellular activity. It is composed of almost nothing but adult fat cells with the usual nerve and blood supply. There are very few blood cells to be found in the parenchyma. Here and there a normoblast is to be seen. A few cells which probably are myelocytes can be found. No giant cells are

noted. There are no definite islands of blood cell formation, simply groups of a few cells scattered through the fat tissue. Practically all types of cells can be found with diligent search, but they are very few. In one place several arteries and nerves can be noted. One of the arteries shows a partially organized thrombus. The red cells in certain instances show a peculiar acidophilic stippling (Fig. 4).

Kidneys: The kidneys show nothing but a few rather fresh surface infarcts, and on microscopic section, a pathologic amount of fat infiltration.

Adrenals: The adrenals are a little dark in color and show a diminished amount of medullary substance, but this is unquestionably partially if not entirely postmortem change.

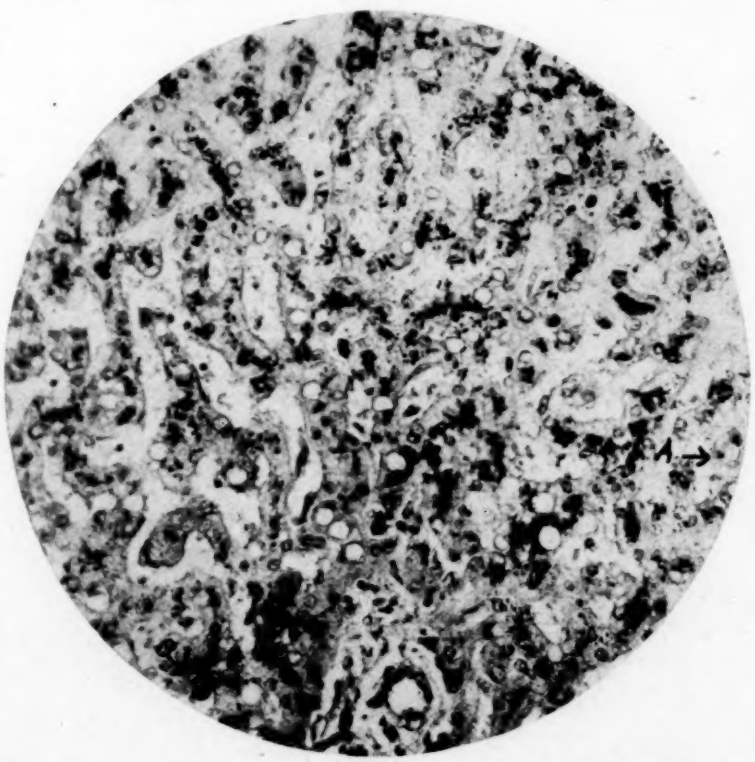

Fig. 1.—Liver. Section illustrates the general distribution of the hemosiderin deposit peripherally in the lobules (Arrow "B") and the amount of central necrosis (Arrow "A").

The other organs, the thyroid, the thymus, the gastro-intestinal tract, the pancreas, the gallbladder and ducts, the bladder, the prostate, the testes, the aorta and the blood vessels, all appear to be perfectly normal. The lymph nodes show relatively the same changes noted in the spleen. The skin shows an increase in pigment both in the basal layer of the epithelium and in the connective tissue chromatophores.

Hematology: The blood cells are interesting. The red cells show achromia, and vary with the intensity of their staining reactions. They show acidophilic stippling for the most part. They have a slight tendency toward macrocytosis. There are no embryonic forms in the circulation and very

few to be found in the marrow. The white cells are peculiarly small, seldom being much larger than the red cells. They are reduced in number. No embryonic forms were found in the circulation, and again, very few in the marrow. Their cytoplasm is very clear, and the characteristic granules are very scarce. No platelets were demonstrated in the sections or in the marrow smears.

PATHOLOGICAL DISCUSSION

Let us now for a moment consider the case with the additional evidence which the pathologic findings give us. The marrow aplasia is so extreme that there is no hesitancy in making the diagnosis of

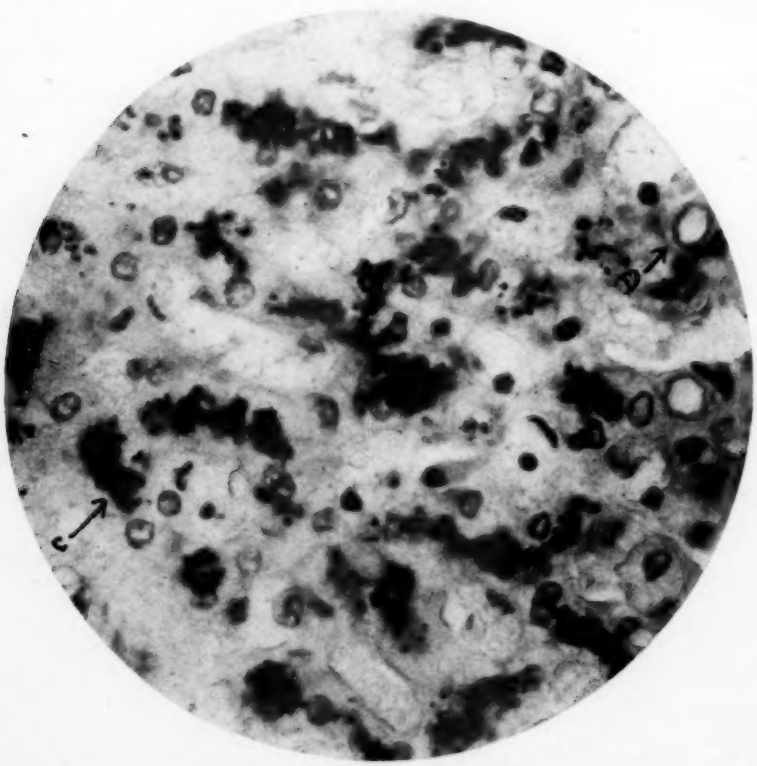

Fig. 2.—Liver. Arrow "C" shows the arrangement of the hemosiderin in the liver cells, and the apparent glycogen deposit in certain of the nuclei (Arrow "D").

aplastic anemia. Now we turn to seek for the cause, if any is to be found, to account for the condition. The immediate cause of death was a failing circulation with dilatation of the heart, accompanied by decompensation. The edema of the lungs was partially responsible as well. The primary cause of death, however, goes back a good deal farther than the failing circulation. For those who do not believe in the primary nature of such an anemia — in its idiopathicity — the

histologic changes in the spleen offer perhaps one solution to the problem. The malpighian corpuscles give very definite evidence of prolonged toxic action such as one sees especially in diphtheria, or in any of the prolonged febrile diseases. It is frequently concomitant with measles. That is perhaps the most obvious answer to the whole story — the prolonged toxic action of the toxin of measles, acting in a cumulative sort of fashion, until the marrow became exhausted. Yet

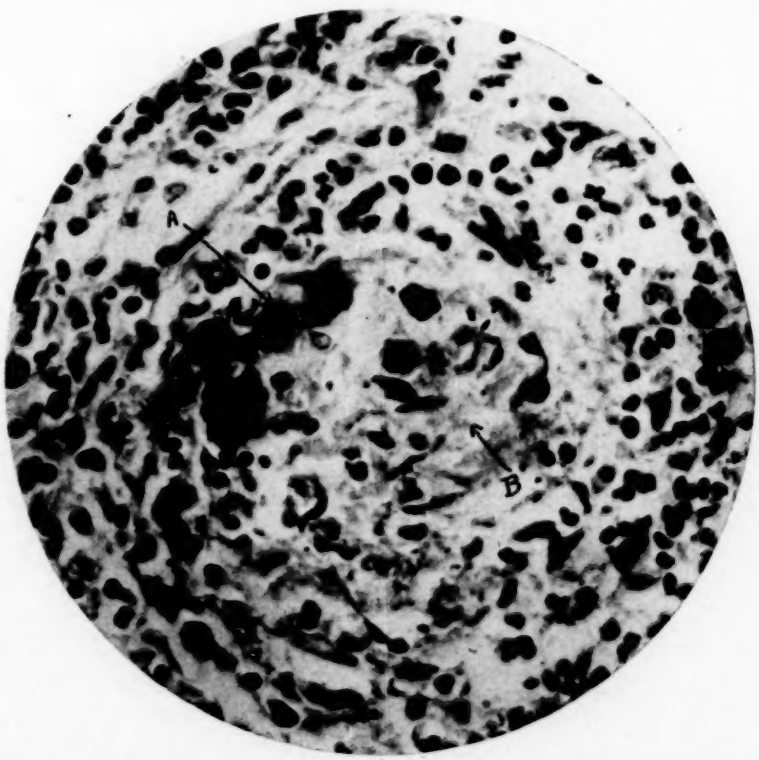

Fig. 3.—Spleen. Section through one of the malpighian corpuscles, illustrating the degree of toxic degeneration which has occurred. Arrow "A" points toward one of the fused endothelial type cells, with several nuclei. Arrow "B" indicates the general necrosis present.

we must remember that the child exhibited no symptoms for practically eight months after he had suffered from his attack of measles, which, incidentally, was a rather light case. Personally, from the literature I have found no such prolonged, cumulative action of the toxin of measles, and most certainly in the relatively few cases of aplastic anemia which have been recorded, measles has not in a single instance played the part of causative agent.

The rest of the pathology gives us absolutely no clue to the cause of the condition. The fatty degeneration of the organs is part of any anemia, merely a question of lack of nutrition. The bone marrow certainly offers no solution to the question. Why? It is merely aplastic, whether primarily or secondarily from exhaustion is impossible to state. That leaves us the liver to consider. There we have a very definite clue in the hemosiderin deposit. In this connection the

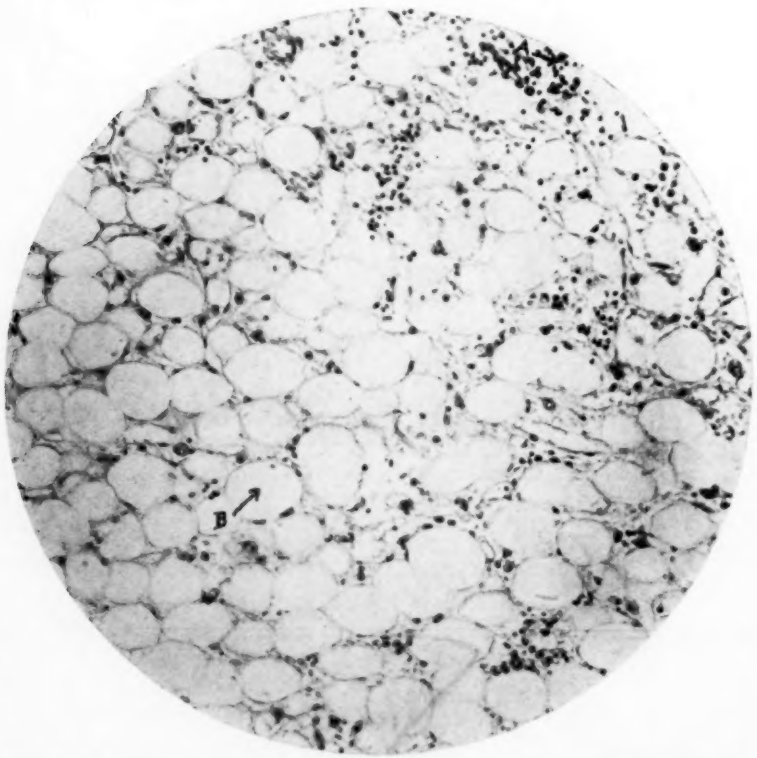

Fig. 4.—Bone marrow from femur. Shows the extreme degree of hypoplasia present. Section is taken from the portion showing greatest activity. Arrow "A" indicates the nearest approach to island formation of the blood cells. This occurs in the vicinity of a large vessel.

argument is advanced that the presence of iron in any great amount is direct evidence of the hemolytic nature of the disease and its consequent inseparability from pernicious anemia. The amount of iron has even been used as a rough index of the severity of the disease in pernicious anemia and of the rate of red cell destruction. In this connection very interesting experimental work has been done in the injecting of hemoglobin and of hemolytic streptococcic serum, but it

brings us no nearer a definite conclusion. As toxic hemocytolysis is one of the foremost theories of aplastic anemia, however, this feature of this case seems to be in direct support of this theory. Opposed to this theory is that in which the very presence of hemosiderin in the liver is used as the basis of a differential diagnosis from pernicious anemia and the other hemolytic anemias. It is to be regretted that the pathology in these cases has been so incomplete, for unquestionably the ultimate classification will be on some sort of pathologic basis.

In this case, however, from the clinical history and from the pathologic findings there was no hesitancy in making the diagnosis of aplastic anemia. An acute aplasia of the marrow with severe secondary anemic changes throughout the organs of the body were indisputable evidences. The case seemed to be of more than passing interest, and as the eventual solution of the problem depends on just such fragmentary bits of evidence, it seemed worth while to present the main features in the hope that they may be of some value in that final solution.

LITERATURE AND GENERAL DISCUSSION

Aplastic anemia is still such an indefinite and vague form of blood disorder that it is interesting to review the literature, and discuss some of the theories which have developed since the condition was first recognized and described by Ehrlich in 1888. Since that time I have been able to find sixty-four cases which have been reported as such. That carries the literature up to December, 1918. Many of those cases are most certainly typical, several others are most questionable, and two or three very definitely present features which seem to exclude them from the classification as it now stands. Many of them present merely the clinical side and have no pathologic findings; these we have to consider very doubtful, as I will show later. Others have very incomplete pathologic findings. That is to be regretted from the scientific point of view, but is of no importance from the diagnostic standpoint.

In 1908 Cabot presented twenty-four cases which he had collected from the literature and deemed authentic. He also mentioned eleven other cases which he rejected from his list as being too atypical or incomplete to be included. This reviews very completely the cases from the time the disease was recognized as a clinical entity by Ehrlich.

In 1911 Hirschfeld presented a new list of forty-four cases which he had collected, which included three of those that Cabot had rejected and twelve which had been included. He also presented a case of his own which was very interesting.

In 1914 Musser published the account of a case which had come under his observation, but which did not come to necropsy for con-

firmation of the diagnosis. He concluded that it followed the clinical course of the disease accurately enough to fall into that category. His work is particularly of value, not so much because of his case, which, unfortunately, cannot be considered perfectly conclusive without necropsy findings, but because of the résumé he gives of the work since 1908, including the more recent theories, and an excellent bibliography. In that same year Heuber came out with another valuable contribution, and he was followed in 1915 with another memorable article by Weber, both with cases and a brief discussion.

In 1917 Minot brought out a paper which has thrown rather a new light on the subject. He makes the rather obvious but previously disregarded similarity of the blood pictures and clinical symptoms of aplastic anemia and purpura hemorrhagica the basis of a new classification. This has opened a new field of thought, and while it may be the clue that will lead to the eventual solution of the problem, at present it certainly tends to make the subject still more confused. Other than these papers, there has been no apparent effort to collect the literature, and one of the chief purposes of this paper is to bring it as nearly up to date as possible. Since 1914, of course, there have been geographical limitations to the current periodicals, but there have been several cases of indisputable diagnosis which have been published. These are appended in the bibliography at the end of this article.

In a paper of this sort there is unfortunately not space enough to allow of a discussion of all the presented cases. A few cases are so flagrantly secondary to some very definite disturbance — infections, pernicious anemia, etc. — that they can scarcely be included among the recorded cases, especially with the diagnosis unconfirmed by necropsy. Among this latter class may be included the cases presented by Blumer, Senator, Wolff, Stitt, Kurpjeweit, Whipple, Musser and Crummer, in none of which are there any necropsy reports. On the other hand, with the clinical picture as definite as it usually is, with no evidence of marrow activity in the peripheral circulation, possibly one is entitled to make the diagnosis of aplastic anemia. Assuredly the mechanism whereby the cells get into the blood stream is interfered with in some way, or the cells are destroyed as fast as they appear in the circulation. In either event the blood picture clinically is the same and very characteristic. I have made a summary of the sixty-two cases which I have been able to find, in regard to their blood pictures, and possibly that would be of some interest.

It is unfortunate that all the data in each case could not be obtained, but incomplete as it of necessity is, it gives a fairly accurate picture of the severity of the disease, its rapid course, and its low red and white counts. In most of the foregoing cases the marrow showed practically complete aplasia.

STATISTICAL ANALYSIS OF CASES OF APLASTIC ANEMIA

Author	Sex	Age	Duration	Hemoglobin, per Cent.	Red Blood Cells	White Blood Cells	Marrow
Acuna	♀	9	1 mo.	..	1.5	4,200	
Aubertin	♂	19	.....	..	0.3	4,900	Fatty
Babonneix and Paisseau	♀	11	.....	25	2.0	leukopenia	Fatty
Barberis	♀	28	.....	..	2.2	682	Fatty
Bloch	♀	63	3½ wk.	28	1.9	3,250	
Bloch	♀	53	2½ wk.				
Blumenthal	♀	42	4 wk.	20	0.9	3,400	Fatty
Baumler	♀	29	3 mo.				
Blumer	♂	50	13 da.	20	1.2	2,000	
Cade	♀	55	1 yr.	..	0.6	40,000	Fatty
Carslaw and Dunn	♂	24	4 mo.	20	1.0	2,000	Fatty
Caussade and Schaeffer	..	..	2 mo.	..	1.0	5,000	Fatty
Chauffard	♂	32	6 yr.?	12	0.4	1,000	Fatty
Crummer	♀	18	2 mo.	17	1.0	1,500	
Deganello	♂	25	2 mo.	..	...	low	Fatty
Dalton	♀	17	2 wk.	..	0.8	3,500	Fatty
Ehrlich	♀	21	1 mo.	..	0.2	1,000	Fatty
Engel	♀	31	3 wk.	18	2.1	3,570	Fatty
Evans and Halton	♂	36	3 mo.	30	1.1	7,200	Fatty
Gulland and Goodall	♂	42	5 mo.	30	1.5	5,000	? among 17 cases of pernicious anemia
Hershberg	♀	16	.....	10	1.4	1,200	
Herz	♂	24	5½ mo.	10	0.3	1,600	Fatty
Herz	♀	32	1½ yr.	10	0.3	2,600	
Hanot	♀	25	2 wk.	..	...	?	Fatty
Hirschfeld	♀	28	9 mo.	..	1.2	3,600	Fatty
Hirschfeld	♀	34	6 wk.	10	2.0	.....	Fatty
Hirschfeld	♂	21	4 wk.				
Heuber	♂	2	.....	15	0.9	7,800	
Krantz	..	..	.....	10	0.3	800	Fatty
Kurpjuweit	♂	68	.....	25	1.1	.....	Fatty
Kurpjuweit	♂	62	6 mo.	25	1.9	600	Fatty
Larrabee	♂	5	1 yr.	25	1.5	5,000	
Lavenson	♀	33	8 wk.	10	0.8	2,500	Ratty
Lucksch and Stefan	♂	62	2½ mo.	..	0.6		
Lesne, Clerc and Loederich	♂	27	6 da.	..	...	2,400	Fatty
Lipowski	♀	31	8 da.	..	...	Normal	Fatty
Massary	♀	21	.....	..	0.7	low	Fatty
Muir	♂	14	2 mo.	12	0.6	7,000	Fatty
Musser	♀	47	2 mo.	21	1.3	2,000	
McWeeney	♂	16	4 da.	37	0.9	250	
Osler	♂	22	12 da.				
Perles	♂	32	4 wk.				
Planchard	♀	29	2 mo.	..	...	Normal	Fatty
Pasteur	♂	24	3 wk.				
Robinson	♀	35	6 da.				
Rheiner	♀	30	3 mo.				
Schaumann	♂	22	8 mo.	..	0.6	.....	? Fatty
Scott	♂	23	19 da.	..	2.0	5,000	Fatty
Steinhaus and Stordeur	♀	43	3 mo.	..	2.0	5,000	Some aplasia
Steinhaus and Stordeur	♂	27	3 mo.	40	1.9	2,700	
Stitt	♂	20	.....	10	0.5	3,000	
Silva	♂	12	15 wk.				
Stone	♂	57—	2 mo.	..	0.3	3,100	Fatty
Schneider	♀	26	1 mo.	18	1.2	1,600	Fatty
Schneider	♀	17	10 da.	15	1.2	900	
Schneider	♀	47	5 mo.	12	0.7	1,400	Fatty
Smith	♂	6	8 mo.	10	0.4	4,000	Fatty
Thomas and Roleston	♂	30	.....	17	0.8	12,000	
Vaquez and Aubertin	♂	19	"yrs."	..	...	6,000	Fatty
Whipple	♂	16	.....	15	0.8	2,200	
Weber	♂	18	2 mo.	12	0.8	2,000	
Zeri	♂	46	3 mo.	18	0.4	2,700	
Zeri	♀	57	6 mo.	..	...	3,500	Fatty
Ziegler	♀	17	1 yr.	35	1.0	2,000	Fatty

SUMMARY

Total number of cases	64
Male	31
Female	30
Unspecified (females probably from history)	3
Average age of 61 cases	29½ years
Youngest	2 years
Oldest	68 years
Average duration—50 cases	3 months
Shortest	4 days
Longest	1½ years
Hemoglobin*—Average of 33 cases	17 per cent.
Highest	35 per cent.
Lowest	10 per cent.
Red blood cells†—Average of 47 cases	1.0
Lowest	0.2
Highest	2.2
White blood cells‡—Average of 44 cases	3,000
Lowest	250
Highest	40,000
White blood cells—Average without 2 highest counts	2,800
Highest	7,800

* The hemoglobin would probably be even lower if all the cases had reported it.
† The red count is given in millions.
‡ The white count would unquestionably be lower if more of the cases had reported them, for a marked leukopenia was mentioned in nearly every case where the count was not given.

Now let us turn for a moment to the theories concerning aplastic anemia. These may be discussed best perhaps under four headings: first, the etiologic; second, the clinical; third, the pathologic, and fourth, the experimental. First, as to the etiology of aplastic anemia, let us acknowledge defeat at the very start. As yet there is nothing but speculation to advance for the cause or causes of the condition. It is essentially a disease of young adults, occurring chiefly in persons between the ages of 18 and 35. The extremes cited are 2 and 68 years of age. Females are perhaps more susceptible than males — the proportion being about equal in this series of cases, however.

There are numerous theories, all equally tenable. Among them may be mentioned the following: first, that it is the most severe form of pernicious anemia; second, that it is secondary to repeated anemia and therefore a secondary anemia; third, that it is secondary to some infectious or toxic disease, the toxin acting primarily on the red cells, causing hemolysis; fourth, that it is a primary aplasia of the marrow by some unexplained toxic action on the marrow itself; fifth, that it is dependent on the platelet element of the blood, acted on again either in the marrow or in the circulating peripheral blood stream.

Pappenheim concludes that it is a secondary anemia, secondary to some primary marrow change (which in turn is secondary to some unexplained and unknown toxic change) and has no relation to the ordinary phanerogenetic anemias, or to the so-called primary pernicious anemia, the result of a primary hemolysis.

Lavenson makes the rather broad statement that aplastic anemia is a variety of pernicious anemia, and that the blood picture is due to hemocytolysis plus inactivity of the marrow, which may be caused by the inhibitive action of the same toxins which produce the hemolysis.

Larrabee after going over the literature both from the clinical and experimental standpoints, in presenting a very unusual case occurring in a boy of 5 concludes that "aplastic anemia is not therefore a disease of itself, but merely a condition which may arise from various causes occurring in secondary anemias, or in pernicious anemia of the destructive type, either as the result of the action of some toxin which destroys the red cells, or from exhaustion of the marrow from over-activity."

Naegli, quoted by Musser, maintains that the relation of aplastic anemia to pernicious anemia is very close, and that the marrow aplasia is due to extraordinarily rapid hemolysis of the red cells, so rapid that the bone marrow cannot keep pace with it and becomes exhausted secondarily. He claims that pernicious anemia is a very constant toxigenetic anemia brought on by toxins from various sources, acting in one of the three following ways: first, by direct hemolysis of the red cells in the peripheral circulation (this has been rather conclusively proved experimentally by McNeil to be unlikely if not impossible); second, by increasing the vulnerability of the red cells to the action of the hemolytic tissues of the body; or third, by stimulating the hemolytic tissues to hyperhemolysis.

Steinhaus and Stordeur, in 1908, published two interesting cases in which everything fits into the usual picture of aplastic anemia except that the marrow was replete with mast cells. They suggest that this may be true in all cases if special stains are used to demonstrate it. This was not found to be so in our case.

Minot lays particular stress on the platelet side of the problem in his comparative studies of aplastic anemia and purpura hemorrhagica, and makes an arbitrary classification of the idiopathic forms of each disease with a large intermediate class which are usually considered as purpura, but which he shows by case evidence to have the pathology of aplastic anemia with the symptoms of purpura. He also differentiates very carefully between the true idiopathic aplastic anemia and the aplasia in other anemias which occurs secondarily to some infection or toxemia, and can more accurately be compared to the experimental aplasia from benzol poisoning. Other points of interest which he makes are those in relation to the clotting of the blood, which in idiopathic aplastic anemia is not very much delayed, but which in coagulating fails to retract in the usual way. His conclusions based on the hemolytic action of the cells in hypotonic salt solution are in general the same as those advanced by McNeil and Hill. The bleeding time is very much increased. This is an important means of differential from cases of hemophilia.

These various views I merely mention to show the difficulties associated with the solving of the problem of etiology.

Fortunately, however, the disease is fairly definite clinically, and not likely to be confused with the other blood disorders, although it may be difficult to differentiate it from purpura hemorrhagica as I have mentioned, or from an aleukemic phase of an acute leukemia, or even from the secondary or pernicious anemias at certain stages in their course. The history will usually clear away those uncertainties. It is characterized by its rapid and progressively fatal course. No cases have been known to recover. Larrabee's case is the only one with a definite remission, and much doubt has been expressed concerning the classification of the case. It has been suggested that it may fall into the group of secondary aplastic anemias following pernicious anemia.

The disease is associated in almost every instance with hemorrhagic manifestations, either as hemoptysis, epistaxis, or purpuric eruption. Often there is fever accompanying it at some period during its course. This may become alarmingly high, but as a rule does not. The skin may or may not show pigmentation, depending a good deal on the rapidity of its course. Another rather constant clinical feature is the cachetic appearance of the patient, who is profoundly anemic and asthenic.

The blood picture is of course the main point of clinical diagnosis. There is usually a tremendous degree of anemia, with the red cells diminished in number to a million or less. The drop may take place so rapidly that within seventy-two hours there may be a fall from two or three million to seven or eight hundred thousand. The hemoglobin is reduced to 10 or 15 per cent. The color index is usually rather low, in contrast to pernicious anemia, although this is not invariably the case, and it may nearly reach one. The white cells are also reduced, and it is interesting to note that it is only the granular leukocytes (the bone marrow derivatives) that are thus diminished, and that there is accordingly a relative lymphocytosis. This is only relative, however, and the count ranges from 800 to 4,000. The differential often shows the polymorphonuclear cells reduced to 8 or 10 per cent., while the lymphocytes make up the rest, frequently being over 90 per cent.

A feature of the disease is a reduction in the number of platelets. The red cells present show comparatively slight changes beyond a certain amount of achromia. There is relatively little poikilocytosis or anisocytosis, although the cells may tend to be macrocytic. There is rarely any stippling or polychromatophilia, or vital stained cells. This is of course accounted for by the lack of marrow activity. The

importance of the blood picture is discussed most conclusively, perhaps, by Minot. The distinguishing features of the blood smear other than these are the absolute lack of normoblasts, megaloblasts, myelocytes, myeloblasts, or any other embryonic form of cell. This picture, conclusive as it may seem, unfortunately can usually be observed at some time or other in a severe case of pernicious anemia, so our final diagnosis must be made at necropsy.

An example of this may be cited briefly by outlining a case which came to necropsy at the Peter Bent Brigham Hospital recently. The patient, a strong, well-developed man of 41, entered the hospital after a gradual increase of rather indefinite symptoms, with hemorrhages, chiefly from the nose. The blood picture gave the impression of an aplastic anemia; the hemoglobin was down to about 25 per cent., the red count about 2,000,000, and the white count a little under 5,000 with a relative lymphocytosis. There were no young forms, nucleated reds, etc., practically no poikilocytosis or anisocytosis, and very little achromia. At necropsy the ribs and upper third of the femur showed unusual hyperplasia of the marrow.

With such a case in mind, many of those presented have to be accepted only with great reservation, and only those with necropsy findings of an aplastic marrow can be accepted with complete confidence. In the aplastic marrow we find a complete replacement of the normal marrow by ordinary fat tissue. Occasionally there may be a fibrous proliferation as well. The marrow may show reddish areas, but these are under the microscope found to be areas of hemorrhage rather than islands of active marrow. The perfectly typical aplastic marrow, such as was found in our case, is a yellowish white fat color, greasy to the knife, and practically completely aplastic — more accurately perhaps, acute hypoplasia, with no true islands of marrow activity, even lymphoidal. Occasional myelocytes and young red cells can be found scattered here and there through a solid mass of adult fat cells, with a stroma of delicate blood vessels and connective tissue. Steinhaus and Stordeur with the *Mastzellen* theory have found no other supporters.

A summary of the reported pathology shows that this aplasia is the most constant and important feature diagnostically. Other features which are usually found are those of any anemia. The organs are pale. There is a diminished amount of fat, and what fat is present, both subcutaneous and in the abdomen, is normal in appearance and not the typical "canary" yellow of pernicious anemia. The mesenteric lymph nodes are very frequently enlarged, the spleen is apt to be hyperplastic. It seldom undergoes any histologic change other than those of congestion and fibrous proliferation. The liver seems to be

a variable factor. One school maintains that the liver is usually of a peculiar golden brown color suggestive of fatty degeneration and hemosiderin deposit. The other school as insistently states that no hemosiderin is deposited, and that that is a valuable diagnostic bit of evidence in differentiating it from pernicious anemia. Personally, I am inclined toward the hemosiderin theory after seeing the tremendous quantities present in this case. Other observers have noted a considerable hemosiderin deposit in the other organs, especially the kidneys and the spleen. Edema of the lungs has been a variable feature, being dependent purely on physical laws. Other changes have shown no general characteristics of any great value diagnostically.

The pathology in this disease has been as unsatisfactory as the clinical classification, and is especially conspicuous in most instances by its absence or incompleteness. Eventually, presumably the classification must be based on a pathologic basis, as the kidney lesions, for example, have had to be, and it is to be regretted that so little advance has been made in the past towards its ultimate solution. That is one of the chief reasons for the existence of this paper — that this fragment may be added to the accumulation of facts from which the final story will be told, and therefore no apologies are offered for the rather minute pathologic findings which have been presented.

On the experimental side little can be added to help solve the problem. Numerous investigators by as many methods have injected various toxins, including saponin, benzol, ricin, hemolytic streptococcic serum, hemoglobin, and many others, and have produced anemias, even marrow aplasia, but there is one fundamental difference which consistently recurs — the fact that with the removal of the cause, if the period of toxicity in the severest cases can be bridged over for a short time, the condition improves, showing that no vital changes have been effected in the marrow in spite of the cellular destruction. Therefore, it is fairer, perhaps, to let these cases fall into the orthoplastic type of anemia — those due to some obvious cause, such as infection, neoplasm, repeated hemorrhage, parasites and the like, rather than into the metaplastic type which pernicious anemia represents. It is unfortunate that the experimental work cannot be gone into at greater length, but it does not fall into the scope of such an article as this. There may be some diversity of opinion in regard to the theory just advanced, but all will at least admit that these experimental cases of anemia fall into one of the two classes cited in which there is definite effort on the part of the marrow to regenerate, and with the removal of the cause the regeneration proceeds, and that therefore these experimental cases cannot be included in the true aplastic anemias. In this connection it is interesting to note two fatal

cases of hemolytic aplastic anemia without the usual accompanying jaundice, resulting from TNT poisoning in the course of the manufacture of munitions.

In spite of all the experimental evidence which has been accumulating in the past few years, and which unquestionably will be the ultimate means of discovering the true etiologic factor of the condition, and in spite of all the clinical findings which after all are fairly similar, no definite cause of the disease has yet been found, and we are rather forced to agree with Larrabee that "aplastic anemia is not therefore a disease of itself, but merely a condition which may arise from various causes."

In conclusion I wish to take this opportunity to thank Dr. John Lovett Morse of the Medical Staff of the Children's Hospital for the privilege of reporting the case and for his interest in it, and Dr. S. Burt Wolbach of the Pathological Department for his invaluable assistance in the study of the pathologic specimens and in the preparation of the photomicrographs.

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THE RELATION OF PARENTAL NATIVITY TO THE INFANT MORTALITY OF NEW YORK STATE

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The New York State Department of Health has for some years past realized the serious menace to the health and economic prosperity of future generations which is involved in the present high rate of infant morbidity and mortality. This is shown by the persistent and successful efforts which the department has made during the last ten years toward reducing the infant mortality of the state. It was believed, nevertheless, at the beginning of the year 1916, that the utmost reduction was still far from being attained, and that, if a more thorough and careful analysis of available statistical data were made much information might be acquired which would aid future efforts in this direction. Accordingly, it was decided to institute such an investigation during that year. The investigation was confined to a study of the data contained in the 1916 certificates of deaths and births, and although in consequence of this limitation it was not as complete as might be desired, the results were in many respects satisfactory. Since the State Department of health has no jurisdiction over that of New York City, no data relating to that city were taken into consideration, the purpose being to study only the conditions prevailing in the territory under its supervision.

Among the several facts elicited which were considered as having a valuable bearing on the subject, the following matter appeared to possess the greatest practical importance.

For the information of the uninitiated, it would be well to state beforehand the technical definition of the term "infant mortality." By infant mortality is meant the number of deaths of infants under 1 year of age per every 1,000 live births which occur in the same area during any one calendar year. It is apparent that in order that rates computed on this basis may be reliable, there should be a complete registration of births and deaths. The registration of deaths in New York State has been known to be virtually complete for a number of years, but it has not been until very recently that the same could be said regarding the registration of births. It may be now stated authoritatively, however, that at present birth registration is so nearly complete in the state of New York that one is justified in accepting the infant mortality rates for the state as exceptionally trustworthy.

The mortality of children under 1 year of age in the state (exclusive of New York City) for the year 1916 was 96 per 1,000 live births. It was the lowest rate ever recorded for this district up to that time. It compares favorably, not only with the rates of most of the great civilized nations, but with those of the majority of the other states of the U. S. Birth Registration Area as well.

TABLE 1.—INFANT MORTALITY OF PRINCIPAL FOREIGN CIVILIZED COUNTRIES AND OF EACH STATE COMPRISING THE U. S. BIRTH REGISTRATION DISTRICT

Section A			Section B		
Year of Last Report	Country	Infant Mortality	Year of Last Report	United States Birth Registration Area, 1915	Infant Mortality
1914	Ireland	87	1916	New York state	96
1914	England and Wales	105	1915	Total U. S. birth registration area	100
1914	Scotland	111		Connecticut	107
1913	Italy	137		Maine	105
1912	German Empire	147		Massachusetts	101
1912	Austria	180		Michigan	86
1912	Hungary	186		Minnesota	70
1909	Russia	248	1915	New Hampshire	110
				Pennsylvania	110
				Rhode Island	120
				Vermont	85

A comparison of the rates in Table 1, coupled with the fact that the New York rate for 1916 represents the lowest point of a curve that has been steadily declining during the past ten years, is a source of much satisfaction to all concerned. That there is room, however, for further improvement may be gathered from the next tabulation (Table 2).

In Table 2, Section A, it will be noted that of the 95.7 infants dying under 1 year of age per each 1,000 births, 46.8 died before the expiration of the first month succeeding birth. Of this number, 33.6 died during the first week and 19.2 before the end of the first day. These rates are considerably above normal, as may be deduced from a comparison of them with the rates for the same age periods recorded for the United States Birth Registration District in 1915 (the latest year for which the figures are available) and with those computed for England and Wales for the year 1914, the last normal year preceding the war. Although the mortality under 1 year was lower in New York by 3.2 and 8.9, respectively, than in the other two districts selected for comparison, it nevertheless had a death rate for the first month higher by 3.1 than that of the United States Registration District, and greater by 8.3 than that of England and Wales. Moreover, these unusually high rates are not due, as may at first be supposed, to any abnormal condition arising during that particular year. Any doubt as to the accuracy of this statement should be quickly dispelled by a glance at Section B of the same tabulation.

TABLE 2.—Infant Mortality in New York State (Exclusive of New York City) in 1916 at Certain Age Periods—Compared with U. S. Birth Registration Area in 1915, and with England and Wales in 1914

	Total Births	Total Deaths Under 1 Year		Deaths Under 1 Day		Deaths Under 1 Week		Deaths Under 1 Month		Deaths Under 3 Months		Deaths Under 6 Months		Deaths Under 9 Months	
		Deaths	Inf. Mort.	Deaths	Inf. Mort.	Deaths	Inf. Mort.	Deaths	Inf. Mort.	Deaths	Inf. Mort.	Deaths	Inf. Mort.	Deaths	Inf. Mort.
Section A															
New York State (exc. of New York City) 1916	103,530	9,912	95.7	1,987	19.2	3,482	33.6	4,846	46.8	6,375	61.6	7,897	76.3	9,016	87.1
U. S. Birth Registration District for 1915*	674,645	67,418	99.9	9,727	14.4	19,907	29.5	29,499	43.7	40,850	60.6	52,332	77.6	60,923	90.3
England and Wales, 1914	879,096	91,971	104.6	.....	.....	.....	.....	33,815	38.5	50,833	57.8	67,335	76.6		
Section B															
Infant mortality in New York State (exc. of New York City) for preceding five years by age periods:															
1915	101,596	10,154	99.9	1,939	19.1	3,480	34.3	4,943	48.7	6,529	64.3	8,156	80.3	9,300	91.5
1914	99,510	10,391	104.0	1,763	17.7	3,447	34.6	4,918	49.4	6,631	66.6	8,354	84.0	9,527	95.7
1913	93,590	11,209	119.8	1,690	18.1	3,335	35.6	4,997	53.4	6,930	74.0	8,913	95.2	10,193	108.9
1912	91,539	10,410	113.7	1,547	16.9	3,150	34.4	4,599	50.2	6,480	70.8	8,251	90.1	9,470	103.5
1911	87,137	10,379	119.1	1,483	17.0	3,043	34.9	4,492	51.6	6,401	73.5	8,163	93.6	9,436	108.3
Total for 5 years	473,372	52,543	111.0	8,422	17.8	16,455	34.7	23,949	50.6	32,971	69.7	41,837	88.4	47,926	101.2

* Includes those states comprising the U. S. Birth Registration District with the exception of New York State outside of New York City.

TABLE 3.—Percentage of Deaths from Principal Causes at Certain Age Periods

Cause	Under 1 Day		Under 1 Week		Under 1 Month		Under 3 Months		Under 6 Months		Under 9 Months		Under 1 Year	
	Deaths	Per Cent.	Deaths	Per Cent.	Deaths	Per Cent.	Deaths	Per Cent.	Deaths	Per Cent.	Deaths	Per Cent.	Deaths	Per Cent.
All causes	1,987	100.0	3,482	100.0	4,846	100.0	6,375	100.0	7,897	100.0	9,016	100.0	9,912	100.0
Measles	.....	.....	1	.....	3	0.1	10	0.2	31	0.4	60	0.7	119	1.2
Whooping cough	.....	.....	.....	.....	8	0.2	69	1.1	114	1.5	165	1.8	205	2.1
Syphilis	14	0.7	19	0.5	55	1.1	118	1.9	159	2.0	174	1.9	182	1.8
Convulsions	13	0.7	45	1.3	69	1.4	85	1.3	96	1.2	103	1.1	108	1.1
Acute bronchitis	.....	.....	9	0.3	54	1.1	119	1.9	161	2.0	196	2.2	228	2.3
Pneumonia	.....	.....	25	0.7	74	1.5	161	2.5	252	3.2	321	3.5	375	3.8
Bronchopneumonia	6	0.3	35	1.0	137	2.8	299	4.7	495	6.3	682	7.6	850	8.6
Diseases of stomach	3	0.1	16	0.4	42	0.9	93	1.5	138	1.8	159	1.8	170	1.7
Diarrhea and enteritis	5	0.3	39	1.1	254	5.2	712	11.2	1,345	17.0	1,797	19.9	2,095	21.1
Malformations	189	9.5	446	12.8	583	12.0	679	10.5	728	9.2	753	8.3	764	7.7
Premature birth	1,199	60.3	1,753	50.4	2,063	42.6	2,110	33.1	2,116	26.8	2,119	23.9	2,120	21.4
Congenital debility	70	3.5	217	6.2	386	8.0	591	9.3	706	8.9	735	8.1	746	7.5
Injuries at birth	297	14.9	469	13.5	503	10.4	506	7.9	509	6.4	509	5.6	509	5.1
External causes	19	1.0	35	1.0	49	1.0	80	1.2	108	1.4	115	1.3	127	1.3
All other causes	172	8.7	373	10.7	566	11.7	743	11.7	939	11.9	1,128	12.5	1,314	13.3

Instead of the mortality rates under 1 month of age being extraordinarily high during 1916, they were in reality the lowest recorded for any similar period during the previous five years. It should be noted, however, that the percentage of reduction since 1911 in the number of these early deaths, did not keep pace with the decline in the rate denoting the total deaths under 1 year. In the latter instance the reduction was 19.6 per cent., but the decrease in the number of deaths under 1 month was only 9.3 per cent., and in those under 1 week only 3.7 per cent. As to the number of deaths under 1 day, there was actually an increase of 13 per cent. It is probable, though, that this increase is due to the greater care and accuracy with which deaths occurring immediately after birth are now reported. Probably a larger proportion of these deaths were formerly reported incorrectly as stillbirths.

In view of the foregoing, it is clear that there are certain causes of infant mortality in operation in this state which up to the present have received too little attention. Else, why should the mortality of infants under 1 year of age have been reduced to a point lower than that of most other large populations, while the death rate under 1 month of age remained above the average? Either we have neglected to give sufficient thought to certain important phases of the matter, or, for some reason, the methods now employed for the reduction of infant mortality fail to reach most babies until after the first month of life. In regard to this question Table 3 should be instructive.

The reader will immediately perceive that of the total number of deaths occurring within the first day after birth, *over 60 per cent. were due to premature birth, 13 per cent. to congenital debility and malformations, and nearly 15 per cent. to injuries at birth, amounting in all to about 88 per cent.* There occurred 1,987 deaths under 1 day, or 20 per cent. of the total under 1 year. The same influences predominated at the end of the first week, accounting for 83 per cent. of the total deaths from all causes. At the end of the first month, 73 per cent. of the entire mortality was due to these causes. From this age onward their relative importance as a factor of mortality decreases rapidly, until, arriving at the age period of 1 year, they were the cause of only 41.7 per cent. of the total number of deaths. On the other hand, the deaths attributable to measles, whooping cough, acute bronchitis, pneumonia, bronchopneumonia, diseases of the stomach, and to infantile diarrhea and enteritis, gradually rose from 11.8 per cent. at the age of 1 month to 40.8 per cent. of those occurring within the first year. If we eliminate from consideration those deaths occurring under 1 month of age, we find that these causes resulted in 68.5 per cent. of the total infant mortality of the next eleven months of life. During the same period the prenatal causes accounted for less than 12 per cent.

Summarizing the foregoing data, we arrive at the following five important points: (1) The infant mortality under 1 year in this State bears favorable comparison with that of most other communities of similar size; (2) this is occasioned by the steady reduction that has been brought about in the infant death rate in New York State during the past decade; (3) this reduction has taken place principally among children over 1 month old; (4) the great majority of the deaths of children above 1 month of age are caused by communicable, respiratory and gastro-intestinal diseases; and (5) about three-fourths of the infantile deaths under 1 month of age are due to prenatal causes. The logical conclusion to be derived from these five facts is that, up to the present we have concentrated our attention so exclusively on the diseases incident to children over 1 month old that we have failed almost entirely to realize the gravity of the situation in regard to those causes having a prenatal origin. It would seem that here is a field requiring the immediate consideration of all interested in the subject of child welfare.

The above facts being established, we may now proceed to another interesting aspect of the subject.

It appears from Section A of Table 4 that the mortality of babies under 1 month old is higher among those born to native mothers than among children born of women of foreign nativity. Although the mortality of children under 1 year of age born of native women was only 87 per 1,000 births compared with 108.4 for children of foreign born mothers, the rate under 1 month for the former was 47.4 as against 45.2 for the latter. Attention is also directed to the fact that the infant mortality of the children of native mothers over 1 month of age and less than 1 year old was only 39.6 contrasted with 63.2 for babies born to foreign mothers.

The extraordinarily high mortality of colored babies is noteworthy, but owing to the small numbers recorded it is doubtful whether these rates may be taken as thoroughly reliable.

By dividing the total foreign born mothers into two main groups, one composed of Italians, Russians, Poles and Austro-Hungarians (which, by the way, comprised about 73 per cent. of the total mothers of foreign birth), and one consisting of all others of foreign nativity, we find that the lower mortality of babies under 1 month of age of foreign parentage is due principally to the first mentioned group (Section B, Table 4). The second group showed even higher rates under 1 month than did the native mothers. Accordingly, it may be inferred that infantile deaths from prenatal causes are particularly numerous among the children of native women, and of foreign women of other than Italian, Russian, Polish or Austro-Hungarian birth. With the latter class, however, one may expect to find the majority of deaths to be caused by communicable, respiratory and gastro-intestinal diseases,

TABLE 4.—Infant Mortality in New York State (Exclusive of New York City) in 1916 According to the Nativity and Color of the Mothers and at Certain Age Periods

Nativity of Mothers*	Total Births	Total Deaths Under 1 Year		Deaths Under 1 Day		Deaths Under 1 Week		Deaths Under 1 Month		Deaths Under 3 Months		Deaths Under 6 Months		Deaths Under 9 Months	
		Deaths	Inf. Mort.	Deaths	Inf. Mort.	Deaths	Inf. Mort.	Deaths	Inf. Mort.	Deaths	Inf. Mort.	Deaths	Inf. Mort.	Deaths	Inf. Mort.
Section A															
Native white	64,889	5,648	87.0	1,313	20.2	2,286	35.2	3,074	47.4	3,968	61.2	4,760	73.4	5,246	80.8
Native colored	696	152	218.4	20	28.7	46	66.1	59	84.8	83	119.3	119	171.0	140	201.1
Total foreign white	37,914	4,112	108.4	654	17.2	1,150	30.3	1,713	45.2	2,324	61.3	3,018	79.6	3,630	95.7
Section B															
Italian, Russian, Austrian, Polish	27,711	3,035	109.5	394	14.2	739	26.6	1,182	42.7	1,638	59.1	2,170	78.3	2,656	95.8
Other foreign white	10,203	1,077	105.6	260	25.5	411	40.3	531	52.0	686	67.2	848	83.1	974	95.5

* There were 31 mothers whose nativity was unknown.

TABLE 5.—Infant Mortality from Principal Causes and by the Nativity of the Mothers—1916

	Total Births	Deaths Under 1 Year from All Causes	Inf. Mort.	Deaths from Principal Causes								All Other Causes	
				Communicable Diseases (1-14 inc.)		Respiratory Diseases (28-29 and 86-98 inc.)		Gastro-Intestinal Diseases (103-104 inc.)		Premature Birth, Congenital Debility and Malformations (150-153 inc.)			
				Deaths	Inf. Mort.	Deaths	Inf. Mort.	Deaths	Inf. Mort.	Deaths	Inf. Mort.	Deaths	Inf. Mort.
Total mothers	103,530	9,912	95.7	471	4.5	1,530	14.8	2,265	21.9	4,435	42.8	1,211	11.7
Total white mothers	102,834*	9,760	94.9	463	4.5	1,505	14.6	2,239	21.8	4,372	42.5	1,181	11.5
Total colored mothers	696	152	218.4	8	11.5	25	35.9	26	37.4	63	90.5	30	43.1
Native white mothers	64,889	5,648	87.0	229	3.5	684	10.5	1,096	16.9	2,943	45.3	696	10.7
Total foreign white mothers	37,914	4,112	108.4	234	6.1	821	21.6	1,143	30.1	1,429	37.7	485	12.8
Italian mothers	12,998	1,259	96.9	90	6.9	313	24.1	317	24.4	402	31.0	137	10.5
Russian mothers (excluding Russian Poland)	3,665	377	102.9	18	4.9	64	17.5	128	34.9	126	34.4	41	11.2
Austro-Hungarian mothers (excluding Austrian Poland)	6,345	822	129.6	41	6.4	165	26.0	273	43.0	268	42.3	75	11.8
Polish mothers (includes German, Austrian and Russian Poland)	4,703	577	122.7	37	7.9	116	24.7	191	40.6	171	36.3	62	13.2
Total for group	27,711	3,035	109.5	186	6.7	658	23.7	909	32.8	967	35.0	315	11.4
British mothers	1,869	134	71.7	6	3.2	14	7.5	22	11.7	68	36.4	24	12.8
Irish mothers	1,879	182	96.9	8	4.2	19	10.1	40	21.3	82	43.7	33	17.6
German mothers	2,296	283	123.3	7	3.0	48	20.9	80	34.8	117	51.0	31	13.5
Canadian mothers	2,219	243	109.5	13	5.9	43	19.4	45	20.2	107	48.2	35	15.8
Other foreign-born mothers	1,940	235	121.1	14	7.2	39	20.1	47	24.2	88	45.4	47	24.2
Total for group	10,203	1,077	105.6	48	4.7	163	16.0	234	22.9	462	45.3	170	16.7

* Thirty-one white mothers, nativity unknown.

since the infant mortality between the ages of 1 month and 1 year for children of these mothers is over 59 per cent. greater than that incident to native mothers' babies of the same ages.

A glance at the next two tables (5 and 6) proves the correctness of the foregoing conclusion.

The striking dissimilarity existing in the relative importance of the several main groups of causes, as distinguished between the native and foreign born mothers, is at once apparent even on a casual review. The infant mortality from communicable diseases was almost 75 per cent. greater among children of foreign mothers than among the babies of native mothers; from respiratory diseases it was over 100 per cent. greater, and from gastro-intestinal diseases the excess was about 78 per cent.; but the rate from prenatal and other causes peculiar to early childhood was higher among the native element by over 20 per cent. Pursuing the analysis still further by dividing the total number of foreign born mothers into the same two groups adopted in the preceding tables, we find these differences much accentuated when the native stock is compared with the group comprising the Italians, Russians, etc. The other foreign-born mothers, considered as a class, also exceeded the native mothers in the death rates from communicable, respiratory and gastro-intestinal diseases, but the rates from prenatal, etc., causes were identical.

Although the table gives us a very fair idea as to the relative prevalence of the several causes among certain classes of the population, it fails to show the percentage of the complete number of deaths from any particular cause that was attributable to each of the three classes of the population.

Table 6 is an attempt to show this relation.

Considering first the deaths of children of native white mothers, it will be noted that although they formed 57.9 per cent. of all the deaths from all causes, they nevertheless contributed only 49.5 per cent. of the total deaths from communicable diseases, 45.4 per cent. of all the deaths from respiratory diseases, and 49 per cent. of the total mortality from gastro-intestinal diseases. At the same time, 67.3 per cent. of all the deaths from premature birth, congenital debility, congenital malformations, accidents at birth, etc., were of children of these mothers. In contrast with these it is interesting to observe that, notwithstanding the fact that only 42.1 per cent. of all the white infant deaths occurred to children of foreign born mothers, 50.5 per cent., 54.6 per cent. and 51 per cent., respectively, of the complete infant mortality from communicable, respiratory and gastro-intestinal diseases were referable to them. As to the mortality from prenatal causes, however, it is to be remarked that only 32.7 per cent. of the total from these causes occurred among this element in the population. This table also shows that the

TABLE 6.—Principal Causes of Deaths of Infants Under 1 Year of Age, Showing Percentages Due to Certain Elements of the Population

	Total Births	Births, per Cent.	Total Deaths Under 1 Year		Causes of Death									
					Communicable Diseases (1-14 inc.)		Respiratory Diseases (28-29 and 86-98 inc.)		Gastro-Intestinal Diseases (103-104 inc.)		Prenatal Causes, etc. (150-153 inc.)		All Other Causes	
			Deaths	Per Cent.	Deaths	Per Cent.	Deaths	Per Cent.	Deaths	Per Cent.	Deaths	Per Cent.	Deaths	Per Cent.
Total white mothers	102,884	100.0	9,760	100.0	463	100.0	1,505	100.0	2,239	100.0	4,372	100.0	1,181	100.0
Native white mothers	64,889	63.1	5,648	57.9	229	49.5	684	45.4	1,096	49.0	2,943	67.3	696	58.9
Total foreign-born mothers	37,914	36.9	4,112	42.1	234	50.5	821	54.6	1,143	51.0	1,429	32.7	485	41.1
Italian, Russian, Austrian and Polish mothers	27,711	27.0	3,035	31.1	186	40.2	658	43.7	909	40.6	967	22.1	315	26.7
Other foreign-born mothers	10,203	9.9	1,077	11.0	48	10.3	163	10.8	234	10.4	462	10.6	170	14.4

high death rates from the first three groups of diseases, incident to the children of the foreign born, were particularly attributable to the Italians, Russians, Poles and Austro-Hungarians.

We may, therefore, consider that our previous surmise has been corroborated, namely, that the chief causes of infant mortality among the native population originate for the most part in adverse prenatal conditions, but that among the foreign element, and especially the Italians, Poles, etc., the most frequent causes of death are communicable, respiratory and gastro-intestinal diseases.

TABLE 7.—INFANT MORTALITY FROM PRINCIPAL CAUSES IN CITIES HAVING POPULATIONS PREDOMINANTLY OF NATIVE STOCK

City	Total Deaths per 1,000 Births	Communicable Diseases	Respiratory Diseases	Gastro-Intestinal Diseases	Prenatal, etc. Causes	All Other Causes	Infant Mortality that Would Have Prevailed if Excess Rates Had Equaled Average Only
Infant mortality for all cities of state, except New York City, 1916...	105	5.0	17.1	26.5	43.4	12.8	
Infant mortality of all Cities listed below.....	100	5.2	15.1	20.7	47.0	12.5	96
Number of	deaths per 1,000 births in excess of the rate incident to all cities						
Syracuse	99	.....	.....	.....	.....	0.7	98
Albany	97	0.3	.....	.....	.....	1.7	95
Binghamton	139	8.9	.....	17.3	7.0	1.8	104
Elmira	96	.....	.....	.....	7.5	5.5	83
Poughkeepsie	117	0.3	.....	.....	17.2	1.7	98
Newburgh	86	0.6	.....	.....	1.1	.....	84
Kingston	94	.....	.....	.....	16.3	.....	78
Oswego	114	0.2	2.1	.....	17.4	.....	94
Lockport	98	0.6	.....	.....	7.0	.....	90
Olean	72	1.0	1.1	.....	4.3	.....	66
Ithaca	88	.....	.....	.....	22.3	.....	66
Middletown	67	.....	.....	.....	0.5	1.2	65
Glensfalls	86	.....	6.0	.....	2.1	3.7	74
Peekskill	81	.....	.....	.....	3.0	.....	78
Watervliet	94	.....	25.9	.....	.....	.....	68
Hornell	86	.....	0.1	.....	4.7	0.9	80
Saratoga Springs	121	.....	33.5	.....	3.5	.....	84
Batavia	107	.....	0.7	.....	18.9	.....	87
Corning	99	.....	.....	.....	23.6	.....	75
Geneva	88	6.3	8.3	.....	.....	1.3	72
Cortland	100	2.4	.....	.....	2.1	2.1	93
Rensselaer	136	13.5	7.6	.....	18.3	.....	97
Fulton	128	5.1	.....	7.3	14.0	0.7	101
Johnstown	44	0.5	.....	.....	.....	.....	43
Oneonta	97	.....	.....	.....	36.9	.....	60
Beacon	87	.....	.....	.....	20.2	.....	67
Plattsburgh	162	4.0	9.9	.....	49.0	9.7	89
Oneida	95	4.5	.....	.....	.....	1.4	89
Port Jervis	117	.....	.....	5.4	25.6	.....	86
Tonawanda	76	.....	.....	.....	16.0	.....	60
Salamanca	92	.....	.....	2.2	8.3	.....	81
Norwich	100	.....	.....	.....	14.5	13.5	72
Mechanicsville	52	.....	.....	.....	3.2	.....	49
Canandaigua	68	.....	.....	.....	2.3	.....	66

With the foregoing established facts in mind, we may naturally expect, when starting a study of infant mortality in any community known to be composed principally of native stock, to find a preponderance of deaths from prematurity, congenital debility, congenital mal-

formations, accidents at birth and other causes peculiar to early childhood; but if the locality under consideration is peopled in large part by Italians, Russians, Poles or Austro-Hungarians, we may reasonably anticipate finding excessive death rates from those diseases having a contagious origin, etc.

We may note that this theory is borne out by the statistics of the individual cities for 1916.

In Table 7 we have a tabulation of those cities which, according to the Federal Census of 1910, were inhabited by an unusually large percentage of people of native stock. Of the 34 cities listed, 25 show collective infant mortalities below 105 (the rate for all the cities of the State, excluding New York City). There are, however, 28 cities showing excessive death rates from prenatal and allied causes. The rate for the total 34 cities from these causes is 47 deaths per 1,000 births as against 43.4 for the whole urban part of New York. Only 4 cities have exceptionally high rates from gastro-intestinal diseases, and only 10 have rates above the average from diseases of a respiratory nature. Attention is directed to the marked difference between the mortality from gastro-intestinal diseases for the total cities here tabulated (20.7) and that of all the cities of the State (26.5).

There is reason to believe that there has been a decided change in the constitution of the population of Binghamton since 1910, on account of improved industrial conditions, etc., and that there is now a much larger proportion of the foreign born resident in that city than there was before the war. If this is true, it undoubtdly helps to account for the unusually high rates from communicable and gastro-intestinal diseases recorded for that city.

In the next table (Table 8) we have selected for study those cities known to have particularly large populations of Italians, Russians, Poles or Austro-Hungarians.

Seven of this group of eight cities had infant mortalities above 105, the total urban rate for the state. The general mortality for the whole group was 115, exceeding the urban rate for the State by 10 in every 1,000 births.

Our proposition appears to receive additional substantiation from the data presented in this tabulation. The rates from communicable, respiratory and gastro-intestinal diseases are all much higher for this group of cities than they are for the cities of the state as a whole, but the rate from prematurity and similar causes is somewhat lower.

The cities listed in the next table (Table 9) are those populated in 1910 by a large proportion of the foreign born other than Italians, Russians, Poles and Austro-Hungarians.

It will be noted that there is no very marked difference between the death rates from the several causes for the cities of this group and for

TABLE 8.—INFANT MORTALITY FROM PRINCIPAL CAUSES IN CITIES WITH EXCEPTIONALLY LARGE POPULATIONS COMPOSED OF ITALIANS, RUSSIANS, AUSTRO-HUNGARIANS AND POLES

City	Total Deaths per 1,000 Births	Communicable Diseases	Respiratory Diseases	Gastro-Intestinal Diseases	Prenatal, etc. Causes	All Other Causes	Infant Mortality that Would Have Prevailed if Excess Rates Had Equaled Average Only
Infant mortality for all cities of state, except New York City.....	105	5.0	17.1	26.5	43.4	12.8	
Infant mortality for group of cities listed below.....	115	8.4	20.3	32.2	41.7	12.4	103
Number of	deaths pe	r 1,000 bir	ths in exc	ess of the	rate incid	ent to all	cities
		(except	ing New Y	ork City)			
Yonkers.....	90	.....	.....	1.5	.....	.....	89
Utica.....	111	.....	7.9	.....	5.1	.....	98
Niagara Falls.....	132	11.0	6.6	12.5	5.1	.....	97
Amsterdam.....	106	4.1	.....	.....	1.3	2.3	98
Lackawanna*.....	190	19.7	28.3	66.5	54.2	20.9	105
North Tonawanda.....	109	5.2	.....	1.4	2.0	.....	100
Little Falls.....	112	1.6	.....	8.7	5.0	.....	97

* Excludes deaths of infants (born of nonresident mothers) in infant home.

TABLE 9.—INFANT MORTALITY FROM PRINCIPAL CAUSES IN CITIES WITH A LARGE NUMBER OF FOREIGNERS BUT NOT PARTICULARLY FROM ITALY, RUSSIA, AUSTRIA-HUNGARY OR POLAND

City	Total Deaths per 1,000 Births	Communicable Diseases	Respiratory Diseases	Gastro-Intestinal Diseases	Prenatal, etc. Causes	All Other Causes	Infant Mortality that Would Have Prevailed if Excess Rates Had Equaled Average Only
Infant mortality for all cities of state, except New York City.....	105	5.0	17.1	26.5	43.4	12.8	
Infant mortality for group of cities listed below.....	101	3.7	16.8	26.8	41.7	12.2	101
Number of	deaths pe	r 1,000 bir	ths in exc	ess of the	rate incid	ent to all	cities
		(except	ing New Y	ork City)			
Buffalo.....	114	.....	3.5	8.4	.....	1.9	100
Rochester.....	86	.....	.....	.....	.....	.....	86
Schenectady.....	79	.....	.....	.....	3.4	.....	76
Troy.....	97	.....	8.3	.....	3.0	0.7	85
Jamestown.....	97	.....	.....	.....	13.4	.....	84
Mt. Vernon.....	89	.....	2.0	.....	1.5	.....	86
New Rochelle.....	70	.....	.....	.....	.....	3.6	66
Auburn.....	84	0.9	.....	1.6	.....	.....	81
Watertown.....	130	2.3	.....	.....	29.6	6.2	92
Cohoes.....	126	2.0	3.9	17.3	2.2	.....	101
Rome.....	101	7.7	.....	4.2	1.2	.....	88
Gloversville.....	144	.....	9.2	11.8	11.6	6.3	105
White Plains.....	69	0.9	.....	.....	3.8	.....	64
Dunkirk.....	109	2.4	.....	1.1	4.5	.....	101
Port Chester.....	65	.....	.....	....	0.2	.....	65
Ogdensburg.....	134	3.9	.....	.....	37.0	.....	93
Hudson.....	157	.....	5.3	21.3	32.3	.....	98
Ossining.....	76	3.9	.....	.....	19.9	0.6	52

those indicative of the mortality in all cities. With the exception of that from gastro-intestinal diseases, they are all slightly lower.

The three preceding tabulations, notwithstanding certain apparent exceptions in the case of a few individual cities, all tend to corroborate the truth of our statement, that an unusually large number of infantile deaths occur annually among the native population of this state from premature birth and congenital causes, while among the foreign born the most important causes of death are those due to insanitary environment, etc. It is very probable that in those cities which seemingly deviate from the general rule there exist certain conditions which tend to distort the usual result. In the first place, it is not unlikely that owing to the extraordinary changes that have taken place in the industrial world since the beginning of the war, the populations of a number of the cities of the state have changed materially in character since the taking of the 1910 census. If this is so, undoubtedly several cities in the foregoing tabulations may have been classed incorrectly. It would be rather difficult, however, to settle this point at present. Another potent factor to be considered is the relative efficiency that has been attained by the local health departments. A highly efficient health officer working in a very unfavorable community might be able to reduce the infant mortality of that district considerably below that of another community in which the natural conditions were comparatively much more favorable to a low death rate, but which was under the supervision of a lax or incompetent health department. The mistake must not be made, therefore, of considering the foregoing three tables as an infallible indication of the theory under discussion. Their general trend, however, is unmistakable.

By showing in these tables only the number of infant deaths in excess of the average, instead of the actual number per 1,000 births that occurred in each city from each cause, we are enabled to discern more clearly the particular problems confronting each individual municipality.

The exceptionally high mortality rates from communicable diseases recorded for the cities of Binghamton, Geneva, Rensselaer, Oneida, Lackawanna, North Tonawanda and Rome are conspicuous.

The unusually large number of deaths from respiratory diseases occurring in Watervliet, Saratoga Springs, Geneva, Rensselaer, Plattsburg, Utica, Niagara Falls, Lackawanna, Troy and Gloversville demands the early attention of the health authorities of those cities.

As to gastro-intestinal diseases, it is apparent that much has yet to be done in order to control this scourge of babyhood in the cities of Binghamton, Fulton, Port Jervis, Niagara Falls, Lackawanna, Little Falls, Buffalo, Cohoes, Rome, Gloversville and Hudson.

The outstanding feature, though, of all three tables, is the almost universal prevalence of a high mortality from prenatal and kindred causes. In forty-eight of the fifty-nine cities tabulated the death rate from these causes was above the average. It clearly indicates the manner in which we have hitherto failed fully to realize the serious nature of this phase of child welfare.

SUMMARY

It is evident that the several causes culminating in the present high mortality of infants are strongly influenced by the customs and racial characteristics of the various peoples inhabiting the state.

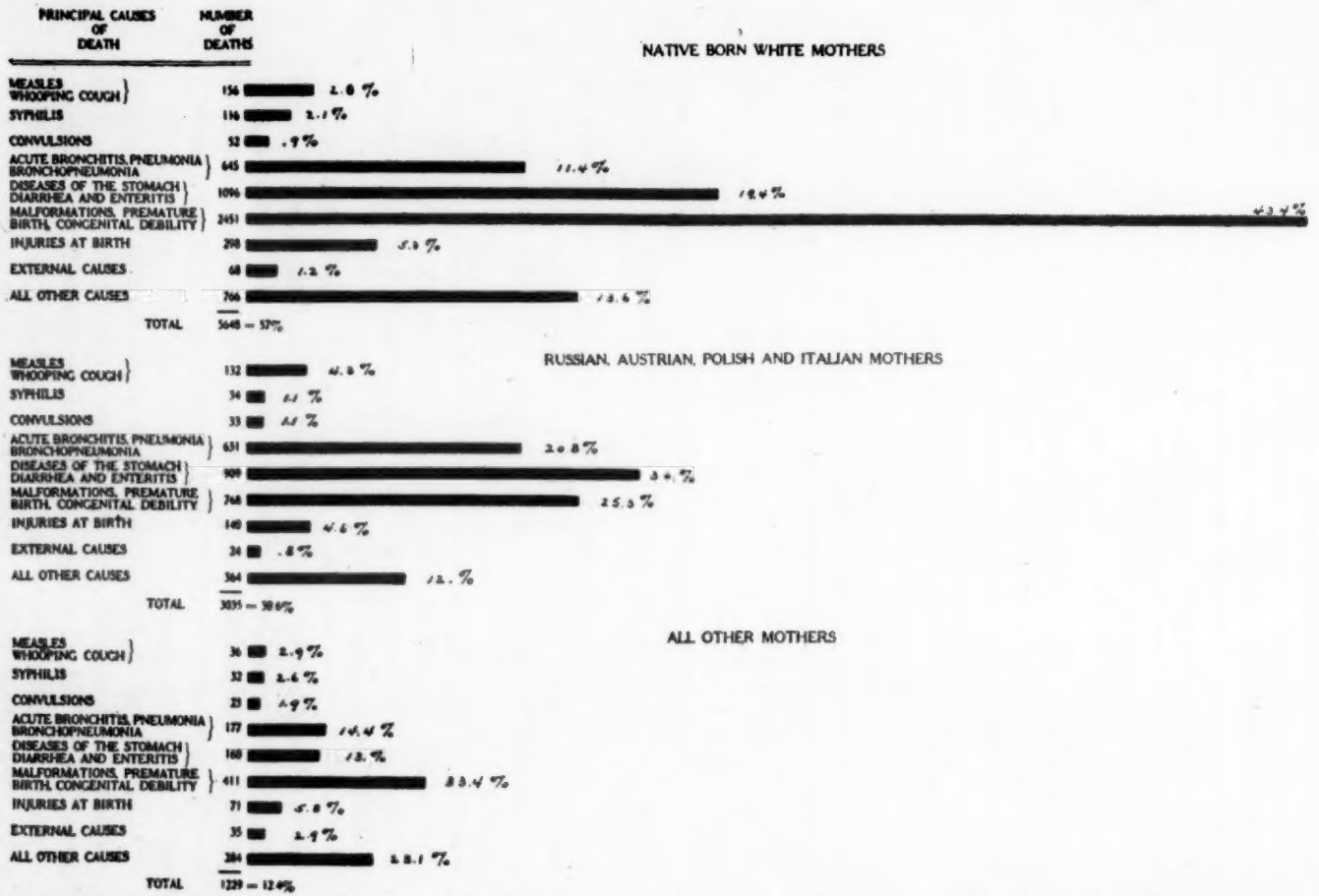
Among the foreign born, and particularly the Italians and Slavic races, the principal diseases are those here classed as communicable, respiratory and gastro-intestinal. Since all these diseases may be said to arise from unfavorable sanitary environment, it is not to be wondered at that they are the dominant causes among the foreign-born population, the majority of whom are poor, illiterate, without knowledge of English and almost wholly ignorant of the elements of modern sanitation, and inhabit, as a rule, the most congested districts of the large manufacturing centers.

The superior environment and intelligence of the average native woman is reflected in the comparatively low mortality of her babies from these diseases.

The fewer deaths from prematurity and congenital defects among children of Italian, Russian, Polish and Austro-Hungarian parents may possibly be accounted for, in part, by the greater vitality and superior constitution of these infants. If this is true, there is the more reason to deplore the excessive mortality incident to them during the later months of infancy, since it would seem that if it were not for the ignorance of their parents, they actually start life with a better chance of survival than do other children.

It is likely, however, that the more general knowledge of contraceptive devices and of artificial methods for the prevention of childbirth together with the more common occurrence of venereal disease and chronic alcoholism among the rest of the population are more directly responsible for the higher infant death rate from prenatal causes within this group.

At any rate, whatever the primary cause may be, the fact still remains that compared with the foreign born, the death rate from prenatal causes is exceptionally high among the children of native mothers, and that it is the main factor to be considered in any campaign for the improvement of child welfare in a community populated exclusively by this element. On the other hand, in a community


PRINCIPAL CAUSES OF DEATH
NUMBER OF DEATHS
NATIVE BORN WHITE MOTHERS
MEASLES WHOOPING COUGH 156 2.8 %
SYPHILIS 116 2.1 %
CONVULSIONS 52 .9 %
ACUTE BRONCHITIS, PNEUMONIA BRONCHOPNEUMONIA 645 11.4 %
DISEASES OF THE STOMACH DIARRHEA AND ENTERITIS 1096 19.4 %
MALFORMATIONS, PREMATURE BIRTH, CONGENITAL DEBILITY 2451 43.4 %
INJURIES AT BIRTH 298 5.3 %
EXTERNAL CAUSES 68 1.2 %
ALL OTHER CAUSES 766 13.6 %
TOTAL 5648 – 57%
RUSSIAN, AUSTRIAN, POLISH AND ITALIAN MOTHERS
MEASLES WHOOPING COUGH 132 4.3 %
SYPHILIS 34 1.1 %
CONVULSIONS 33 1.1 %
ACUTE BRONCHITIS, PNEUMONIA BRONCHOPNEUMONIA 631 20.8 %
DISEASES OF THE STOMACH DIARRHEA AND ENTERITIS 909 30. %
MALFORMATIONS, PREMATURE BIRTH, CONGENITAL DEBILITY 768 25.3 %
INJURIES AT BIRTH 140 4.6 %
EXTERNAL CAUSES 24 .8 %
ALL OTHER CAUSES 364 12. %
TOTAL 3035 – 30.6%
ALL OTHER MOTHERS
MEASLES WHOOPING COUGH 36 2.9 %
SYPHILIS 32 2.6 %
CONVULSIONS 23 1.9 %
ACUTE BRONCHITIS, PNEUMONIA BRONCHOPNEUMONIA 177 14.4 %
DISEASES OF THE STOMACH DIARRHEA AND ENTERITIS 168 13. %
MALFORMATIONS, PREMATURE BIRTH, CONGENITAL DEBILITY 411 33.4 %
INJURIES AT BIRTH 71 5.8 %
EXTERNAL CAUSES 35 2.9 %
ALL OTHER CAUSES 284 23.1 %
TOTAL 1229 – 12.4%

Deaths of infants under 1 year in New York State (exclusive of New York City) during 1916, from principal causes and by the nativity of the mothers.

TABLE 10.—A Comparison of Infant Mortality in the State of New York (Exclusive of New York City) and in the U. S. Birth Registration Area During 1916

Nationality of Mother	Total Infant Mortality		Communicable* Diseases		Respiratory Diseases (28-29 and 86-89 inc.)		Gastro-Intestinal Diseases (150-153 inc.)		Premature Birth, etc. (150-153 inc.)		All Other Causes	
	New York	U. S. Regist.† Area	New York	U. S. Regist.† Area	New York	U. S. Regist.† Area	New York	U. S. Regist.† Area	New York	U. S. Regist.† Area	New York	U. S. Regist.† Area
Total white mothers	94.9	**99.0**	4.5	**5.2**	14.6	**16.0**	21.8	**25.1**	42.5	**40.0**	11.5	**12.7**
Native white mothers	87.0	**89.8**	3.5	**4.3**	10.5	**11.4**	16.9	**19.2**	45.3	**43.1**	10.7	**11.7**
Total foreign-born white mothers	108.4	**107.9**	6.1	**6.1**	21.6	**21.6**	30.1	**32.0**	37.7	**34.9**	12.8	**13.4**
Italian mothers	96.9	**99.0**	6.9	**7.0**	24.1	**26.7**	24.4	**23.7**	31.0	**28.4**	10.5	**13.1**
Russian, Austro-Hungarian and Polish mothers	120.7	**115.8**	6.5	**6.0**	23.5	**22.0**	40.2	**40.3**	38.4	**33.8**	12.1	**13.7**
British mothers	71.7	**86.6**	3.2	**4.1**	7.5	**12.5**	11.7	**17.4**	36.4	**40.6**	12.8	**12.0**
Irish mothers	96.9	**103.1**	4.2	**4.0**	10.1	**15.6**	21.3	**25.6**	43.7	**45.3**	17.6	**12.6**
German mothers	123.3	**103.5**	3.0	**3.9**	20.9	**16.4**	34.8	**31.3**	51.0	**38.7**	13.5	**13.1**
Canadian mothers	109.5	**121.2**	5.9	**7.4**	19.4	**18.3**	20.2	**29.1**	48.2	**51.7**	15.8	**14.7**
Other foreign-born mothers	121.1	**100.2**	7.2	**6.2**	20.1	**19.3**	24.2	**28.3**	45.4	**33.7**	24.2	**12.6**

* New York State rates based on causes "1 to 14 inc." (International List of Causes of Death).
U. S. Registration rates based on causes 3, 5, 6, 7, 8, 9, 11, 14, 15, 16, 17, 18, 19 (International List of Causes of Death).
† Includes the whole state of New York, together with all of New England, Pennsylvania, Michigan and Minnesota.

composed largely of Italians or Slavs, the principal causes to be combated are those arising from poverty, ignorance, overcrowding and imperfect sanitation.

In all future measures for the conservation or betterment of child life, closer attention should be given to the foregoing factors than has heretofore been the case. Otherwise, we are likely not only to fail to carry our project to the most successful issue, but it is possible that, if disregarded, all our efforts may be futile. A careful study of the character and habits of the population should be the first step taken in any attempt to improve child life. On the result of this study should be based the whole course of the campaign. If the population is discovered to be preponderately native, the proper course to be adopted should be mainly one of education in regard to prenatal conditions. If it is found to be largely of foreign born stock, it will be necessary to plan principally for a course of instruction in the proper feeding and care of the infant and for the improvement of sanitation. This procedure would seem to be both rational and practicable, and should be the basis of all future campaigns for infant welfare that aim at efficiency and success.

APPENDIX

Since the foregoing article was written there has been published by the Bureau of the Census of the United States Department of Commerce a report on birth statistics for the United States Birth Registration Area for 1916.

This report includes a number of interesting tables relating to the infant mortality for the Birth Registration Area. Among them, Table 5 (Page 83) has particular significance, owing to its application to the subject of this article. Table 10 is a reproduction of this table in an abridged form, showing rates instead of actual numbers, and so constructed as to facilitate a comparison of its matter with parallel data bearing on the State of New York.

The similarity of the rates of the two areas is so conspicuous that elaborate analysis is needless.

The reader will immediately perceive that although the native element of the population in each instance has a lower infant mortality due to *all* causes than has the foreign element, the native population, nevertheless, shows much higher rates from the prenatal causes. Furthermore, despite the fact that infant mortality from communicable, respiratory, gastro-intestinal, and "other causes" is generally lower in New York State than in the Registration Area, the infant death rate from premature birth, etc., is appreciably higher in the former than in the latter district.

Other noticeable features are, the especially high death rates of Italian children from respiratory diseases, the excessive mortality of

Russian, Polish and Austro-Hungarian children from gastro-intestinal disorders and the exceptionally large number of deaths of babies born to Canadian mothers due to prenatal causes.

The striking disparity noticeable between the general infant mortalities of Canadian children born in the State of New York and of those born in the Registration Area, may probably be accounted for by the fact that in New York a smaller proportion of the Canadian population is of French stock than is the case in the larger area. Since the infant mortality of the French-Canadians is considerably higher[1] than those of English origin, the larger proportion of them inhabiting the United States Birth Registration Area would have a tendency to increase the infant mortality of the total Canadian population of that area.

Likewise, the difference in the rates of the "Other foreign born" group is probably due to the larger proportion of Scandinavians included in this division of the population in the Birth Registration Area than happens to be the case in New York State.[2] The Scandinavians having a very low infant death rate, undoubtedly tend materially to lower the infant mortality of the "Other foreign born" group of the United States Birth Registration District below that of New York.

As to the difference in the rates of the German population, there seems at present to be no satisfactory explanation.

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1. According to the U. S. Census of 1910 the French Canadians constituted 39 per cent. of the total Canadian population of the states that now compose the U. S. Birth Registration Area, while in the state of New York (exclusive of New York City) they formed only 22 per cent. of the total Canadian population.

2. By the same census we find that in the U. S. Birth Registration Area about 52 per cent. of the population which we have listed as "Other foreign born" was composed of Scandinavians, while only 38 per cent. of the same group in New York State (outside of New York City) were of this race.

A SIMPLE METHOD OF MODIFYING THE FAT CONTENT OF MILK FOR INFANT FEEDING*

AMY L. DANIELS AND HONORA ENGLISH
MADISON, WIS.

The more frequent use of low fat milk formulas, especially in certain cases of infant feeding, makes necessary some simple method of modifying the milk in homes where the fat content of the milk is unknown or a variable factor. Even when formulas furnishing larger methods of fat are desired, an easy and reliable method of modification, applicable to home conditions, will be helpful.

There are already in general use several methods suited to meet the conditions for which they were devised. For example, if a high fat milk formula is desired, Holt's[1] method of siphoning off the upper part of the milk is in every way adequate; or, if one desires a 3 per cent. milk and has a 4, 4.5 or 5 per cent. milk, Schereschewsky's[2] method may be employed, although according to Darling[3] this has certain limitations, for the results are apt to be inconstant, and it is not easy to measure thirds of ounces with the usual household apparatus. It also fails to meet all conditions, for no provisions are made for obtaining very low fat formulas or for modifying 3 or 3.5 per cent. milk.

For general purposes, Darling's[3] method of modifying the fat content of milk more nearly meets the conditions desired. Given a 4 per cent. milk, it is possible to secure mixtures containing approximately 2, 3 or 5 per cent. milk fat. The milk is allowed to stand in the bottle until the cream line is distinct. Then the upper 6 ounces of cream are dipped off, leaving 26 ounces of milk. Such milk contains, according to Darling, 1 per cent. of milk fat. To obtain a milk containing 2 per cent. of fat, one-third of the cream which has been removed is restored to the skimmed milk. If a 3 per cent. milk is desired two-thirds of the cream is restored. This method apparently fulfills the need when a 4 per cent. milk is used as the basis of modification, but it is not suited to conditions in the average home, since the milk usually falls considerably below the 4 per cent. standard.

Any household method of milk modification will be found to vary somewhat from time to time, depending on the skill of the individual

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* From the Department of Home Economics, University of Wisconsin.

1. Holt, L. E.: Diseases of Infancy and Childhood.
2. Schereschewsky, J.: U. S. P. H. and M.-H. S. Hygienic Laboratory Bull. 56, p. 726, 1910.
3. Darling, E. W.: Boston M. and S. J. **165**:747, 1911.

and the condition of the milk to be modified. With ordinary care, however, it is possible by using the method herein described to obtain milk containing 2, 3 or 4 per cent. fat as desired, from a supply containing an unknown amount of fat.

The method consists in allowing the milk to stand on ice until the cream line is distinct — about ten or twelve hours. The cream is then carefully removed, so that as little mixing as possible takes place. For this purpose a Walker-Gordon dipper with a removable bottom has been found most convenient. In order to obtain milk containing 2 per cent. fat, 2 ounces,[4] or 54 c.c., of the removed cream should be added to 24 ounces of the skimmed milk; for a 3 per cent. milk 4 ounces, or 108 c.c., should be returned to 24 ounces of the skimmed milk; and for a 4 per cent. milk 6 ounces, or 162 c.c, should be added.

The length of time the milk has stood after bottling is of considerable importance in securing good results with the method, for milk which has stood a too short period contains a varying amount of fat in the skimmed portion. The results of tests on milk skimmed after standing from 6 to 8, 10 to 14, and 24 hours, respectively, are given in Table 1.

Milks which had stood on ice from ten to fourteen hours had distinct cream lines, and the percentage of fat in the samples of skimmed milk tested were comparatively uniform, averaging 0.8 per cent. regardless of the fat content of the original milks. Pasteurized milk, containing 3.6 per cent. of fat, was found after skimming to contain practically the same amount of fat as raw skimmed milk which contained originally 4.2 per cent., or 4.6 per cent. of fat. Milk which has stood twenty-four hours averaged only slightly less fat than that which had stood from ten to fourteen hours. On the other hand, milk which had stood only six or eight hours had no distinct cream line, and the percentage of fat in the skimmed portion was considerably higher than the milk which had stood longer, the pasteurized milk containing from 1 to 1.2 per cent., and the raw from 1.4 to 1.8 per cent. fat. In order to obtain the best results with our method, the milk should be allowed to stand on ice the shortest possible time that will yield a skimmed milk having a consistently low fat content. In from ten to fourteen hours there is a distinct cream line which facilitates separation of the cream and makes possible a fairly uniform skimmed milk.

In determining the percentage of fat in our cream mixtures, the skimmed milk (24 ounces) was thoroughly mixed with the desired amount of cream, 2, 4 or 6 ounces,[4] and an aliquot part tested by

4. The cream was measured in a container holding 1 ounce of milk, or 27 c.c. of cream.

TABLE 1.—Relation of Time to Cream Formation

Exp. No.	Dairy	Kind of Milk	Fat Content of Milk, per Cent.	Time of Standing, Hours	Fat Content of Skimmed Milk, per Cent.
1	K	Pasteurized[5]	3.6	10-14	0.8
2	K	Pasteurized	...	10-14	0.8
3	K	Pasteurized	...	10-14	0.8
4	K	Pasteurized	...	10-14	0.6
5	K	Pasteurized	...	10-14	0.8
6	K	Pasteurized	...	10-14	1.0
7	K	Pasteurized	...	10-14	0.8
8	K	Pasteurized	...	10-14	0.8
9	K	Pasteurized	...	10-14	0.8
10	S	Pasteurized[5]	2.8	10-14	1.0
11	S	Pasteurized	3.0	10-14	0.8
12	H	Raw	4.2	10-14	0.8
13	H	Raw	...	10-14	0.6
14	H	Raw	4.2	10-14	0.8
15	H	Raw	...	10-14	0.8
16	H	Raw	...	10-14	0.8
17	H	Raw	...	10-14	0.8
18	H	Raw	...	10-14	0.8
19	H	Raw	...	10-14	0.9
21	H	Raw	...	10-14	0.6
22	H	Raw	...	10-14	0.8
23	U	Raw	4.6	10-14	1.0
24	U	Raw	...	10-14	0.9
25	U	Raw	...	10-14	1.0
26	C	Raw	4.0	10-14	0.8
27	C	Raw	4.0	11-14	0.8
28	Z	Pasteurized[6]	3.8	6-8	1.2
29	Z	Pasteurized	...	6-8	1.0
30	Z	Pasteurized	...	6-8	1.2
31	C	Raw	4.0	6-8	1.4
32	C	Raw	4.0	6-8	1.6
33	C	Raw	4.0	6-8	1.8
34	C	Raw	...	6-8	1.4
35	U	Raw	4.8	6-8	1.6
36	U	Raw	...	6-8	1.4
37	U	Raw	...	6-8	1.4
38	U	Raw	...	6-8	1.4

5. Pasteurized by the "flash" method.
6. Pasteurized by the "hold" or "vat" method.

TABLE 1.—Relation of Time to Cream Formation—(*Continued*)

Exp. No.	Dairy	Kind of Milk	Fat Content of Milk, per Cent.	Time of Standing, Hours	Fat Content of Skimmed Milk, per Cent.
39	C	Raw	4.0	24	0.8
40	C	Raw	...	24	0.8
41	C	Raw	...	24	0.6
42	U	Raw	4.6	24	0.8
43	U	Raw	...	24	0.8
44	U	Raw	...	24	0.6
45	Z	Pasteurized	3.8	24	0.6

TABLE 2.—Modification of Skim Milk by the Addition of Cream

(Series 1)

Exp. No.	Skim Milk, Ounces	Fat Content, Per Cent.	Per Cent. of Fat Resulting from Cream Addition		
			54 C.c.	108 C.c.	162 C.c.
1	24	0.8	2.0	3.0	4.0
2	24	0.8	...	3.1	4.2
3	24	0.8	2.0	3.1	4.2
4	24	0.8	...	...	4.0
5	24	0.6	1.9	3.0	4.0
6	24	0.5	2.0	3.0	4.0
7	24	...	2.0	3.0	4.0
8	24	0.8	2.0	3.0	4.0
9	24	...	2.0	3.0	4.0
10	24	...	2.0	3.2	4.2
11	24	0.8	2.0	3.0	4.0
12	24	0.6	1.8	3.0	4.0
13	24	0.6	2.0	3.0	4.0
14	24	0.7	1.9	3.0	4.1
15	24	0.8	2.0	3.1	4.0
16	24	0.5	1.8	3.1	4.0
17	24	0.8	2.0	3.0	4.0
18	24	0.9	2.1	3.0	4.0
19	24	1.0	2.0	3.1	4.0
20	24	1.0	2.1	3.1	4.1

means of the Babcock method, duplicate tests being made in all cases. From time to time the fat content of the milk as it was received from the dairy was also tested.

In order that conditions might be as comparable as possible, in the first series of tests the skimmed milk was weighed and the cream to be added measured in graduates, 54 c.c., 108 c.c. and 162 c.c. being equivalent to approximately 2, 4 and 6 ounces, respectively. The data of the trials are summarized in Table 2.

From Table 2 it may be seen that the method, when used under carefully controlled conditions, produces fairly uniform results, the variations being within 0.1 or 0.2 per cent. of the desired standard. The criticism, however, may justly be made that in the household conditions cannot be so carefully controlled. Finely adjusted balances and graduates, so freely used in the chemical laboratory, do not form a part of the usual household equipment. What are the limits of error when the ordinary household appliances are used? Summarized in Table 3 are the results obtained when the skimmed milk was measured in a standard 8-ounce measuring cup, and the cream in a Chapin dipper delivering 27 c.c.

TABLE 3.—Modification of Skim Milk by Addition of Cream

(Series 2)

Exp. No.	Skim Milk, Ounces*	Fat Content, Per Cent.	Per Cent. of Fat Resulting from Cream Additions		
			2 Chapin Dippers	4 Chapin Dippers	6 Chapin Dippers
1	24	0.8	2.0	3.0	4.0
2	24	0.8	2.0	3.0	4.0
3	24	0.8	...	3.1	4.1
4	24	0.8	1.9	2.8	3.8
5	24	0.6	2.0	3.0	4.0
6	24	0.8	2.0	3.0	4.0
7	24	...	2.0	3.0	4.0
8	24	0.8	2.0	3.2	4.2
9	24	0.5	1.8	2.9	3.9
10	24	0.6	2.0	3.1	
11	24	0.6	1.8	3.0	4.0
12	24	0.8	...	3.0	
13	24	0.8	...	3.0	
14	24	0.9	2.0		
15	24	0.8	2.0		

* Measured in an 8-ounce measuring cup.

Equally accurate results may be obtained when household utensils are employed as when carefully calibrated chemical apparatus is used. In selecting the household utensils, however, care should be taken that these fulfill the requirements, for there is some variation in the amount which so-called "standard" measuring cups hold. For this reason it would be better to use a graduate for measuring the milk. The standard Chapin dippers have been found to vary only slightly; other cream dippers may deliver as low as 20 c.c., and therefore should not be used indiscriminately.

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